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EDITORIAL

SALTATORY CONDUCTION

The concept of saltatory transmission of activity in nerve fibres was first stated as long ago as 1925,[1] and has been shown to be correct by experimental investigation. The basis of saltatory conduction is that there is discontinuous propagation of the nervous impulse from one excitable structure to the next across a passively conducting length of nerve fibre. Three essential features of the mechanism, which have had to be proved experimentally, are: (*a*) the excitable structures are only present at the nodes of Ranvier, (*b*) the internodal parts of nerve fibre passively conduct impulses without any excitability, and (*c*) the nervous activity is propagated from one node to the next through local electrical circuits.[2]

By experiments such as unipolar (cathode) stimulation of a single myelinated nerve fibre at various points along its length, or by bipolar stimulation within one internode, evidence is obtained that excitable structures in myelinated fibres are discontinuous and in fact situated at the nodes. It has also been proved that not only excitability but also activity is confined to the nodes. By recording action potentials with a microelectrode at different sites around an active node it is shown that it is the node which produces the electrical field, and that the shape of the action potential in concentric circles around the node is always the same. Narcotics have no influence on myelinated nerve fibres when applied to the internodal portion but the nodes are affected by them and conduction is blocked if more than one node is rendered inexcitable. Blocking of conduction by cold is also not observed unless the nodes are cooled. Cooling of the internode does not block the conduction but does reduce the amplitude of the longitudinal current by increasing the longitudinal resistance of the axis cylinder. Solutions of various kinds, including local anaesthetics, have no action when applied to the internode in concentrations that affect the nodes.

The electrical activity observed along the internodal circuits of the myelinated nerve fibre shows characteristic discontinuities. As a consequence of the activity of the

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SALTATORIESE GELEIDING

Die onderbreking van aktiwiteitsgeleiding in die senuweevesels is 'n begrip wat so ver terug as 1925 [1] gekonstateer is en dit is proefondervindelik gestaaf. Die grondslag van hierdie begrip is dat senuweeprikkelgeleiding van een prikkelbare struktuur na die ander oor 'n afstand van passief geleidende senuweeweefsel onderbreek word. Drie essensiële eienskappe van die meganisme moes eksperimenteel bewys word, t.w. (*a*) die prikkelbare strukture word slegs by die knope van Ranvier gevind, (*b*) die dele van die senuweevesels wat tussen die knope lê dien, sonder enige prikkeling, as passiewe prikkelgeleiers, en (*c*) die senuweeaktiwiteit word van een knoop na die ander deur plaaslike elektrisiteitsbane [2] gelei.

Eksperimente soos die eenrigtingsprikkeling (katode) op verskeie plekke langs die lengte van 'n murgskedesenuweevesel, of die tweerigtingsprikkeling tussen die knope, het bewys dat die prikkelbare strukture van murgskedevesels nie onafgebroke is nie, en dat hul inderdaad by die knope gevind word. Dit is ook bewys dat prikkelbaarheid sowel as aktiwiteit tot die knope beperk is. Deur mikro-elektrodelesings te neem van die spannings op verskeie plekke rondom die aktiewe knoop is dit bewys dat die knoop die elektrisiteitsveld skep en dat die spanning altyd in konsentriese sirkels rondom die knoop voorkom. Verdowingsmiddels wat tussen die knope toegedien word het geen uitwerking op murgskede-senuweevesels nie maar wel op die knope en as meer as een knoop onprikkelbaar gemaak is, word geleiding versper. Koue kan nie geleiding versper nie tensy die knope verkoel is. Verkoeling tussen die knope versper nie geleiding nie maar dit verminder die sterkte van die krag deur weerstand langs die akson te verhoog. Konsentrasies van verskillende soort oplossings, plaaslike verdowingsmiddels inkluis, wat op die knope 'n uitwerking het, het geen uitwerking tussen die knope nie.

Die elektrisiteitsaktiwiteit langs die tussenknoopstroombane van die murgskede-senuweevesels toon kenmerkende onderbrekings aan. As gevolg van die aktiwiteit van die knope moet elektrisiteitstekens in

nodes the electrical signs must be considered in terms of currents and potentials. The longitudinal action currents present discontinuity with respect to time, and also shape. In the case of action potentials discontinuities can be observed with respect to space (shape) but not with respect to time owing to the retarding function of the myelin sheath.

There is also evidence that saltatory conduction occurs in the fibres of the central nervous system, where there are nodes in the myelinated fibres as in peripheral myelinated nerve fibres. These have been demonstrated histologically as interruptions of the myelin sheath at regular intervals though less widely spaced than in the peripheral fibres.[3] At these nodal points the fibres are easily excitable but midway between them they are inexcitable.

Even with the electron microscope it has not yet proved possible to discover the structure of the nodes. The axis cylinder is constricted at the nodes to about 50% of its internodal diameter. The gap which is not covered by myelin is usually not more than 0·5μ and never more than 1 μ in length.

While it has been convincingly demonstrated that the excitable and active region of myelinated fibres is confined to the nodal area, so far no one has seen any membrane structure in this region; nevertheless the term 'nodal plasma membrane' is used to describe this active structure.

terme van strome en spannings beskryf word. Daar is onderbreking van die aktiwiteitstrome langs die akson beide wat tyd en distansie betref. In die geval van aktiwiteitspanning is onderbrekings ten opsigte van distansies, maar nie van tyd nie, genoteer, en dit is aan die vertragingsfunksie van die murgskede te danke.

Daar is ook rede om aan te neem dat onderbreking van geleiding in die vesels van die sentrale senuweestel plaasvind waar knope in die murgskedevesels aanwesig is soos in die geval van die periferale senuweemurgskedevesels. Histologiese demonstrasies toon onderbrekings van die murgskede op gereelde afstande aan alhoewel die afstande nie so ver uitmekaar is nie soos in die geval van periferale vesels.[3] By hierdie knooppunte kan die vesels maklik geprikkel word maar halfpad tussen die knope is hulle nie prikkelbaar nie.

Selfs met behulp van die elektronmikroskoop kon die struktuur van die knope tot nog toe nie ontbloot word nie. Die deursnit van die akson is ongeveer 50% kleiner by die knoop as tussen die knope. Die gaping wat nie deur die murgskede bedek is nie, is gewoonlik nie meer as 0·5μ en nooit meer as 1μ nie.

Alhoewel die prikkelbare en aktiewe streek van die murgskedevesels sonder enige twyfel tot die knoopgebied beperk is, het niemand tot nog toe enige membraanstruktuur in die gebied waargeneem nie; maar desnieteenstaande word hierdie aktiewe struktuur as die „knoopplasma-membraan" beskryf.

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'MIST. EXPECT. SED.'

Cough mixtures are among the most frequently prescribed medicines [1] and are the last stronghold of polypharmacy. Yet the choice of cough remedy often seems to lack a scientific basis. 'Expectorant' drugs are said to increase bronchial secretion. Some text-books [2] increase confusion by calling them 'sedative expectorants', a term used by others for opiates, which decrease the frequency of coughing. Recent articles refer to this second group as 'cough suppressants' or 'antitussive agents', which seem preferable names.

Dunlop [1, 2] found that mixtures containing expectorants plus cough suppressants are prescribed at least as often as either type alone. Although illogical, this does not appear to be dangerous, for the only controlled experiments [4] with ammonium salts, potassium iodide, ipecacuanha and senega in human subjects with natural coughs found these expectorant drugs ineffective at official dosage. Indeed, several authorities recommend a hot cup of tea [5, 6] as more efficient. It is not denied that reflex expectorants taken on an empty stomach in nauseating doses will increase bronchial secretion or that potassium iodide will do the same in a patient with idiosyncrasy.[5] Important points in assessing cough medicines are the natural history of the disease (ordinary acute bronchitis usually subsides in a few days on its own) and parallel treatments (bed rest, etc.).

The opiate-content of many double-action cough mixtures is low—often no more than $\frac{1}{4}$ the dose of morphine recommended for cough in the British National Formulary.[5] Little remains but the possibility of placebo effect—a saline injection can reduce coughing [7]—but it seems inconsiderate to make the patient pay proprietary prices for this benefit.

Where respiratory inflammation is evoking sputum which is being adequately coughed up, there is little point in trying to increase the amount of mucus or reduce coughing. The logical management of such a cough is to treat the pneumonia, tuberculosis, heart failure, etc., to smoke less or, in coryza, the *vis medicatrix naturae*.

Probably the only sound indication for a 'cough medicine' is to use a cough suppressant for cough in excess of sputum. All the proved cough suppressants belong to the morphine group. Heroin is the most powerful, but a very dangerous drug.[2] Morphine and codeine are the most commonly used. The dose of morphine recommended for cough suppression is between 1·5 and 2·0 mg.,[5, 8] that for codeine between 15 and 60 mg. Over this range there seems little to choose between them; their cough-suppressant, analgesic, cerebral-depressant and other effects are of the same order. Above these doses codeine shows no increase in any of

these effects,[2,7] while the effects of morphine steadily climb, and mere cough suppression can eventually become respiratory arrest. In practice morphine is generally used in Camphorated Tincture of Opium and mixed with expectorant drugs which would be emetic if the antitussive dose were much exceeded, while codeine is used alone as a syrup. This seems a sensible way of using them, but codeine does sometimes lead to addiction.[2, 8]

Of the newer morphine relatives, methadone ('physeptone') may be used for cough in the same dose as morphine. Its antitussive effect [7] and its dangers approximate to those of morphine. Another product, dextromethorphan, appears to have a similar cough-suppressing effect to codeine.[9]. It has not yet shown side-effects, central depression or addiction liability.

Although central depressants of other chemical type do not have antitussive action in sub-anaesthetic dosage,[7] it has not been clear whether the antitussive and narcotic actions of the morphine group are fully separable. However, this year there have been reports from two sources that nalorphine (n-allylnormorphine—'Lethidrone'), which has been introduced as a specific antagonist to morphine, is as good a cough suppressant as codeine in animals [10] and man.[11] It was given orally in the same dose as codeine. Perhaps this drug—which has not been found toxic except to morphine addicts—will be the ideal suppressant for ordinary dry coughs.

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INFLUENZA TODAY AND TOMORROW*

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I propose in this lecture to discuss rather briefly the present state of knowledge about influenza, dwelling especially on those areas where the interests of the clinician and the laboratory worker overlap. Then I shall discuss in more detail recent studies on the epidemiology of the disease, referring especially to the evidence which suggests that the virus is constantly changing.

Clinical Diagnosis

From a purely clinical point of view influenza is very hard to distinguish from other acute upper respiratory infections. The most useful guide is that cases of true 'flu tend to come all together in a rush. Sporadic cases which may not be very different from them clinically are usually not due to an influenza virus. Influenza outbreaks rarely occur two years running; so people who say they are afflicted with influenza every year (or even oftener) almost certainly suffer from something else. There is a disease which may simulate influenza both clinically and from its occurrence in outbreaks. In 1937 my colleagues and I called it febrile catarrh (Stuart-Harris *et al*, 1938). Recently in America it has been called acute undifferentiated upper respiratory disease—A.R.D. for short. We prefer febrile catarrh both because it is the older name and because we tend to suffer from epidemics of initials as well as of influenza.

Influenza, as I shall discuss in a moment, can be definitely diagnosed by means of laboratory tests. Febrile catarrhs have tended to consist of what remains and is not 'flu, something not precisely diagnosable. They comprise the numerous cases of exudative tonsillitis

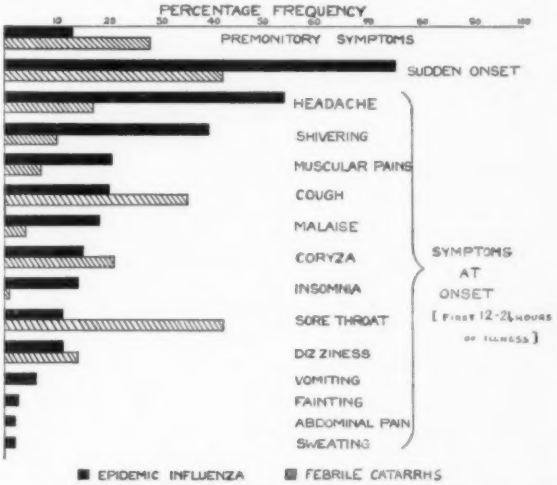

Fig. 1. Symptoms in (1) epidemic influenza and (2) febrile catarrhs (with acknowledgements to *The Practitioner*).

which are not due to streptococci and they also grade into primary atypical pneumonia. In 1938 Stuart-Harris, working with us at Hampstead, tried to distinguish between febrile catarrh and influenza clinically, relying on the symptoms presenting at the onset (Fig. 1). At

* A paper based on lectures delivered by Dr. Andrewes as Nuffield Lecturer visiting the Universities of Cape Town, Witwatersrand, Pretoria and Natal during October and November 1954.

the end, he felt that he could diagnose with some confidence whether an *outbreak* was due to influenza or not, but he could not be sure of placing an individual case.

The A.D. Viruses

Quite lately there have arisen hopes of making the diagnosis of febrile catarrh more precise. As you are aware, tissue culture has come into much prominence lately in poliomyelitis research. Cultivation of human embryonic and other tissues offers a promising method of study for those viruses which will not grow in fertile eggs nor infect convenient experimental animals. One can hope to assay such viruses by detecting destructive change (cytopathic effects) on cells in the cultures. Hilleman and Werner (1954) have cultivated from throat washings of people with febrile catarrh or primary atypical pneumonia, viruses which produce cytopathic effects in a strain of epithelial cells (HeLa cells) derived from a human cervical cancer. These viruses appear to be virtually the same as others described a little earlier by Rowe, Heubner and Ward (1953); these were obtained by cultivating adenoids removed at operation; they produced cytopathic effects in other cultures of adenoids, in HeLa cells and other cultures, and were called AD or adenoid degeneration viruses. My colleagues at the Common Cold Unit at Salisbury in England (Dr. Pereira and Miss Chaproniere) have obtained similar agents from adenoids. The American workers have found that these viruses produce few or no symptoms in human volunteers inoculated intranasally; we have just begun to study them seriously at Salisbury. They may prove of much epidemiological interest. In Britain, outbreaks of disease in the febrile-catarrh category are apt to occur when young people, especially recruits in the armed forces or school-children reassembling at boarding schools after the holidays, are suddenly intimately mixed up together. One wonders whether relatively avirulent agents may not thus find an opportunity to acquire virulence. The AD viruses are already known to be of several serological types as shown by neutralization and complement-fixation tests. Before long we may be in a position to attribute outbreaks or individual cases of respiratory infection to one or other of the viruses of this group. They may well prove to be a family of viruses easily transcending in importance the Coxsackie viruses. Their causative relation to febrile catarrhs has not however been definitely established as yet.

Laboratory Diagnosis of Influenza

Far more definite than all this is the fact that we can diagnose virus influenza in the laboratory with some confidence. Three methods are available.

(i) One can obtain virus from throat-washings of patients in the first few days of the disease by inoculating these into the amniotic cavity of fertile eggs. Virus present in high titre in the embryonic fluids of infected eggs is absorbed on to and will agglutinate the red blood cells of various species. Virus can be readily detected in an inoculated egg by finding that these fluids have acquired such agglutinating power. The specificity of the agglutination is checked by discovering whether it is inhibited by antisera against the known strains of influenza.

(ii) This agglutinin-inhibitor is the basis of the second diagnostic test. This is easier to carry out but cannot give an answer so early in the disease. One requires 2 serum samples from the patient, one taken as early as possible, the other 10 days or more after the onset. If the patient has been infected with influenza, the second sample should show a considerable increase in power to inhibit agglutination of red cells by preparations of a standard strain of the virus. Two samples are necessary because most normal human sera show some haemagglutinin-inhibiting antibody, and a rise in titre must be demonstrated to establish that infection has occurred.

(iii) The third test is that of complement-fixation, and this has two great advantages. In between outbreaks most normal sera have little or no complement-fixing antibody for influenza. As with the previous test, two samples are desirable for diagnosis, but with complement-fixation they are not essential. A single sample often suffices for presumptive diagnosis. Then there is the question of multiplicity of strains of virus. There are 3 influenza viruses: A, the most important cause of major outbreaks; B, causing lesser outbreaks, mostly very local ones; and the lately described C, the importance of which in medicine is still in doubt. The complement-fixation test distinguishes very sharply between these 3 and so one can soon say whether a patient has A, B or C or none of them. But the haemagglutinin-inhibition test reveals greater complexities. Influenza virus A is of various antigenic subtypes: if one tests one's convalescent sera against the wrong subtype one may be misled as to whether one is dealing with influenza A or not. The test is deceptively easy to perform but full of pitfalls for the unwary.

Antigenic Changes and Epidemiology

This leads me to my next point. The antigenic complexities and vagaries of the virus are a curse to the clinical pathologist who wants to know: is this influenza A or not? But they are the breath of life to the man who wants to unravel the fascinating epidemiological mysteries of influenza. For they enable him to distinguish one brand of 'flu from another and to trace its wanderings over the earth. It has become clear during the last few years that influenza viruses are constantly changing in their antigenic make-up: the term 'antigenic drift' has been used. Fig. 2 shows diagrammatically what is believed to be happening. Influenza outbreaks tend to occur every other year and the antigenic make-up

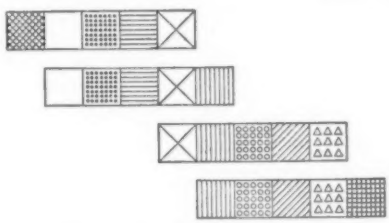

Fig. 2. Diagrammatic representation of 'antigenic drift' in influenza of successive epidemics (by permission of H.M. Stationery Office).

of strains is apt to shift a little from one outbreak to another; very occasionally a major change occurs. This instability of influenza A is of obvious advantage from the virus's point of view. After an epidemic, the herd immunity to the virus is high and only wanes slowly. If a rather new virus emerges it has a greater chance to get going than the old type, for herd immunity against it will be less complete. Once it does get going, it can spread very fast and widely and dominates history for a while. This may help to explain why old strains of 'flu tend to die out and why we do not have a lot of important types going around the world at the same time.

Influenza between Outbreaks

We still do not understand what happens to influenza between outbreaks. In one month there may be in a country tens of thousands of people with influenza and there may be hundreds of millions of virus particles in each of them—say 10^{14} particles altogether. A couple of months later it may be impossible to find an influenza virus in that country, outside a laboratory. The death rate amongst these viruses has been appalling—but incidentally there has been an opportunity for Natural Selection to act in a colossal way to foster a suitable mutant. Some think that as an epidemic subsides, the virus' activities pass on to some other country with lower herd immunity and so on round and round the world. Others think that the virus goes 'underground'—metaphorically speaking—into a place and form in which we cannot identify it, waiting to emerge modified or unmodified to start an epidemic two years later or when the time is ripe. This may merely mean that it keeps ticking over very quickly causing subclinical cases and a very occasional sporadic clinical case, never drawing attention to its presence. We shall see later that there is evidence for both undetected endemicity *and* for occasional trans-oceanic spread.

THE WORLD INFLUENZA CENTRE

In 1948 an organization was set up by WHO for the study of influenza on a world-wide basis. It had become increasingly apparent that the antigenic changes which influenza viruses underwent would make much more sense if one looked at what was happening in the world as a whole. For example, in 1946 a variant strain of influenza appeared in Australia; it was not thought to be of special interest, but in 1947 similar strains were recognised all over North America and much of Europe. The World Influenza Centre (WIC) is now at the National Institute for Medical Research in Mill Hill in the north of London; it keeps in close touch with central laboratories in North America and with designated WHO influenza laboratories in most countries in the world. At present there are 2 in South Africa, Cape Town and Johannesburg. These WHO laboratories keep the WIC informed as to local activity of influenza, and send in the strains they isolate, so that these can be compared with those isolated from other countries. The sort of information coming in is best appreciated by study of maps depicting the happenings in 3 successive epidemics, occurring at 2-year intervals.

In 1948-49 influenza appeared in Sardinia in early autumn, and apparently did so simultaneously in a number of villages spaced quite far apart, as if some factor had activated latent virus over a fairly wide area. Soon after, it was reported from Sicily and from the mainland of Italy, and thence spread to most of western Europe—Switzerland, France, northern Spain, west Germany and the Low Countries, reaching the English Channel about the New Year. It took a little while—till March—to become epidemic in England. It also spread to Denmark, probably by sea, and to Iceland. It apparently failed to pass the Iron Curtain—we had quite good contacts at that time with Czechoslovakia and Hungary; possibly rather free commercial and social intercourse is necessary to allow an influenza virus to spread effectively. Viruses sent to us from all over western Europe were serologically indentical; so it is pretty certain that one virus strain was concerned (Chu, Andrewes and Gledhill, 1950). It looked as if influenza was actually spreading in epidemic form; it is, however, possibly that a seeding of one strain of virus had occurred some time ahead of the epidemic and that it had remained inapparent until successively activated at a later date by changes in climatic or other conditions.

There was little or no influenza in Europe during the following winter, but in May 1950 there were localized outbreaks in Sweden. It had happened before that local 'incidents' of this sort in early summer had preceded a big epidemic in the following winter, so the Swedes were encouraged to keep a special eye on what happened during the ensuing months. Nothing however was noticed till October 1950, when influenza appeared both in Denmark and Sweden. The strain concerned—we later called it the Scandinavian strain—was identical with that of the small precursor outbreak of the previous May, and a little different from that of the 1948-49 epidemic. Influenza turned up shortly after in Holland and Germany and about Christmas-time in eastern England. Then it appeared around Newcastle and other ports handling much traffic across the North Sea, a week or two earlier than in the rest of Britain. Then we had influenza in Liverpool and the neighbourhood, of a very vicious nature, killing old and chronically ill people in considerable numbers. This, Liverpool, strain was antigenically different from the Scandinavian one. Both strains turned up in different parts of England; for some unknown reason, however, it was only around Liverpool itself that mortality was particularly high. From the continent, apart from the north, we obtained viruses of the Liverpool type, especially from those along the Mediterranean, Spain, France, Italy, Turkey and Israel. We were soon most interested to discover that viruses which had been sent to us some months earlier from South Africa, both Johannesburg and Cape Town, and one or two from Australia, were also of the Liverpool type (Isaacs, Gledhill and Andrewes, 1952). The suggestion was inevitable that this type of virus had originated in the southern hemisphere, in the southern winter, and had migrated across the equator to give trouble a little later in the ensuing northern winter. There were some reports of influenza in between whiles in French West Africa, but there is no certainty that these were really due to virus A. It is an interesting possibility that influenza, unlike migratory birds which follow the

sun, actually follows the winter by moving at the appropriate season across the equator. An objection to this theory in its simplest form is of course that we do not have influenza in either hemisphere, every winter.

To return to 1951, the Liverpool strain of influenza apparently crossed the Atlantic without difficulty to produce influenza in America before the end of the winter.

Again we in Europe were fairly free from 'flu in the 1951-52 winter, but in 1952-53 it attacked us again. In contrast to the 2 previous epidemics, it did not seem to arise from any particular focus, but was reported almost simultaneously from several European countries, from North America and Japan; and in all these areas it was of the same general type, much like the 1950-51 Scandinavian virus. In a few countries, notably Portugal, viruses recovered were all or mainly of the old Liverpool type; so far as we know Portugal had not been attacked by this virus in 1950-51. North America, which had had the Liverpool type in 1950-51, had the Scandinavian one in 1952-53. Thus in most countries the Scandinavian antigen, which seems to be of more recent development than the Liverpool one, is seen as replacing the older one in the epidemic strains of virus (Andrewes, 1953; Isaacs and Archetti, 1954).

In 1946 a new Australian virus arose and populated the Northern hemisphere; in 1950 the Liverpool type turned up, also apparently in the southern hemisphere, and similarly passed in a wave over much of the face of the earth. The Scandinavian type has recently been recovered from Australia, so you in the south have apparently not got the monopoly of inventing new influenza viruses. So far, however, all the recent South African A viruses we have received have continued to be of the Liverpool type. Apparently the Scandinavian variety has not yet obtained a foot-hold here. Maybe you have that one still coming to you. Or perhaps we shall get a new type in Europe in the coming winter and you will in due course receive that one and miss out the Scandinavian one in the series. There are practical conclusions to be drawn from such studies as these. We heard, last June, of influenza having broken out in several Union Castle liners and of influenza cases and contacts coming ashore from these ships at Southampton. Should we, in Britain, have to worry about the possibility of an outbreak of influenza in consequence? There were two reasons for refusing to panic. First, influenza rarely gets going in the summer time (the experience of 1918 forms, of course, a tragic exception). Second, the viruses isolated were all of the Liverpool type. Herd immunity against a previously experienced strain is usually good for some time; so we felt that the Liverpool type of 'flu was unlikely to obtain a foothold in Britain during the summer; and events have so far justified our confidence.

Thinking along these lines, I am tempted to speculate largely in terms of time and space. If, in a well-mixed-up community, there is a gradual antigenic drift in the influenza viruses, the people will never meet a wholly foreign virus, only a partly new one having much antigenic overlap with strains encountered earlier, and they will therefore not be afflicted overwhelmingly. But if such drifts took place in two communities isolated from each other, the changes in the virus might follow different courses, so that when, after some years, the communities met again, both of them might meet in consequence changed influenza viruses widely differing from those against which they had immunity. It is even possible that if, in South Africa, only the Liverpool type persists for some years, while the virus undergoes progressive changes elsewhere in the world, you may, when a foreign strain does reach you, find it has diverged a good deal and found you particularly vulnerable just because it is so different from your old Liverpool acquaintance. Studies on these lines are in their early stages and insufficiently precise for my words to cause any serious alarm. On the whole I think the moral to be drawn is a very cheerful one: if transport by air and other means tends to make the whole world one, there is less and less danger of the breeding of new brands of 'flu in remote corners of the earth and of pandemics caused by their breaking loose. I do not think even Iron Curtains would be an effective barrier over a long period: indeed we have evidence that 'flu strains like those of the western world have been occurring recently beyond the curtain.

VACCINES

Besides throwing light on influenza epidemiology, study of antigenic changes amongst 'flu viruses are of practical importance for vaccine production. Some well-known influenza A strains, like WS and PR8, are easy to work with and high-titred products can be readily made. But those strains have ceased to be of practical importance in living epidemiology; we want to know how to protect against the virus of this year's—and, better still, of the next epidemic. Clearly, we can judge much better what strains to include in a vaccine, when we can forecast accurately how new strains are moving on the face of the earth. We have lately concluded that a wise policy is to include in a vaccine a representative of the most recent strain which has shown itself capable of causing spreading epidemics, together with some of the last-but-one spreading strain. Then when a new one turns up, we include that and discard the earlier of the 2 strains already incorporated. Thus, the manufacturer will only have to change strains as regards half his production at one time. I referred to new strains capable of causing spreading epidemics because at the World Influenza Centre we have recently received from distant parts of the world viruses quite distinct antigenically from other current strains. These have at times been isolated examples, and we have heard no more of them. Maybe they never had a future and are nature's evolutionary failures. Or perhaps we shall hear more of them later on.

It is not enough to put into a 'flu vaccine a really modern up-to-date strain. Influenza viruses differ in many properties and not all will make good vaccines. Some refuse to grow in high enough titre. Others show other disadvantages which only become apparent when the manufacturer gets down to large scale production. One has to choose a 'good' strain.

My colleague, Dr. Isaacs, has lately obtained evidence that influenza viruses which have been adapted to grow in the lungs of mice are much better antigens in mice

than are egg-grown viruses. There is further some evidence that they are also better antigens in man. We are carrying out in Britain this winter a vaccine trial from which we hope to obtain more information on this point.

Another question concerns the use of oily adjuvants in virus vaccines. Addition of mineral oil and emulsifying agents to poliomyelitis and influenza vaccines has been found to increase their efficacy considerably, as judged by laboratory tests. Antibody production is slower in starting, but titres rise considerably higher and remain high for much longer (Salk and Laurent, 1952). Of all this there is no doubt; but some people receiving these oily vaccines have developed nodules at the site of inoculation, often coming on after some months and sometimes turning into sterile abscesses. Even though this happens rarely, it would be satisfactory to know how to avoid it; and it therefore seems better to delay very extensive use of such adjuvant vaccines in the meantime.

I will conclude with a few general comments on the use of virus-vaccines against influenza.

Since most people have a basic immunity to influenza, 'flu vaccines can be regarded as essentially a booster or secondary antigenic stimulus. That is why, very fortunately, one seems to get as good results from one dose as from 3.

Immunity after such a dose probably lasts for several months, so that a dose given a month or so before the usual epidemic season is likely to give the best result. In a country which tends to experience an outbreak every other year, it is useless to vaccinate in an 'off-year'; a dose every other year should suffice.

You may want to know whether influenza vaccines are any good anyway. I think they are some good but not nearly good enough. In the most favourable trials they have cut down the incidence of 'flu to about a quarter of that in a control group. A recent trial in Britain showed a reduction of about 40%. But all trials are very hard to assess. So often we find mixed with virus influenza a lot of febrile catarrhs against which one cannot expect the vaccine to protect. One can assess the results in terms of serologically proven 'flu and not on a purely clinical basis, but there are fallacies either way. It is probably impossible to assess a vaccine adequately except in the presence of an epidemic which is almost pure influenza A.

There is also an economic aspect to the whole thing. Influenza can, and often does, cause serious paralysis to the economy of a country, with much resulting economic loss. If one could protect against that with a vaccine causing lasting immunity, it would be undoubtedly worth while. But, as I have told you, things are not so simple and manufacturers may not be enthusiastic about making a vaccine of frequently changing composition, involving from time to time the scrapping of big stocks of out-dated products. It is not practical to try to immunize millions of people against influenza, every other year. It may be practical to have vaccine available for smaller numbers of people in key positions. In any case, I am sure it is worth while to learn all we can about vaccinating against influenza. We hope never again to have a lethal pandemic like that of 1918-19, but we cannot be over-confident; and should such a catastrophe recur, there would certainly be enormous demands for vaccine.

There is, almost unexploited, an entirely different approach to the question: the possibility of vaccinating with a live attenuated virus. In an emergency one could produce such a living virus, which would only need to be given in small doses, far more quickly and cheaply than is possible with formolized vaccines; in fact, it might be the only feasible way of vaccinating very large numbers quickly. Influenza A virus is so labile and adaptable that it should, in theory, be one of the easiest to modify in the required direction. In fact, I have lately found that a neurotropic variant (the NWS strain) can be given intranasally to ferrets without producing fever or symptoms, while inducing solid immunity to the original non-neurotropic WS virus. A difficulty is, however, likely to arise in that in a population resistance varies so much from one individual to another. One subject may be able to stop dead an attenuated virus so that it never gets far enough to stimulate his immunity, while for another the same virus may not be attenuated enough and may produce mild symptoms.

Vaccines, however, are not everything. I hope that full understanding of the epidemiology of influenza may, in the end, show us how to control it by attacking some vulnerable link in its rather complex epidemiological cycle.

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NAPT FOURTH COMMONWEALTH HEALTH AND TUBERCULOSIS CONFERENCE, LONDON

The subjects for discussion at the main sessions of the Fourth Commonwealth Health and Tuberculosis Conference to be held by the British National Association for the Prevention of Tuberculosis at the Royal Festival Hall, London, on 21-25 June 1955, will be as follows:

'The Preventive Outlook To-day.'

'Child Hygiene and Infection.'

'Tuberculosis—A Problem of Different Races.'

'Do Death Rates Matter?'

'X-ray and Tuberculin Surveys—Their Meaning and Interpretation.'

'Choice of Drugs in Medical and Surgical Treatment of Tuberculosis.'

'The Psychology of the Patient and Relatives as a Factor in Successful Treatment.'

Authorities on tuberculosis from countries all over the world will be amongst the speakers. Discussions and clinical meetings will also be held and time will be given for practical demonstrations, visits to sanitoria, hospitals and clinics.

ANEURISMA VAN DIE ARTERIA LIENALIS

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Aneurisma van die arteria lienalis is 'n seldsame verskynsel en dit kom maar min voor dat die toestand vóór operasie gediagnoseer word. Owens en Coffey (1953) meld in 'n besonder volledige oorsig van die letterkunde dat bloot 14 uit 'n totaal van 198 gevalle vanaf 1770 wat hulle versamel het, vóór operasie gediagnoseer is. Ons voel dus dat 'n beskrywing van 'n geval van aneurisma van die arteria lienalis wat vóór operasie tentatief gediagnoseer is, publikasie regverdig.

LETTERKUNDE

Owens en Coffey het die letterkunde só noukeurig en volledig in oorsig geneem vanaf die beskrywing van die eerste geval deur Beaussier in 1770 dat alleen interessante feite uit hulle uitstekende artikel aangehaal sal word.

Ongetwyfeld die mees dramatiese geval, wat hulle beskryf, is dié van James A. Garfield, President van die Verenigde State van Amerika, wat twee maande nadat hy deur 'n sluipmoordenaar se koeël in die buik getref is (1881), aan 'n gebarste aneurisma van die arteria lienalis beswyk het. Die aneurisma was hier die gevolg van die koeëlbesering.

Winkler (1903) word deur hulle aangewys as die eerste persoon wat die letsel in 'n lewende pasiënt waargeneem het. Hy het terloops daarop afgekom gedurende 'n laparotomie op 'n 25-jarige verpleegster. Hy het die arteria lienalis afgebind, en die milt verwyder maar die aneurisma gelaat. Die pasiënt het herstel en vir 25 jaar na die operasie in goeie gesondheid geleef.

Hoegler (1920) was die eerste persoon wat die diagnose van aneurisma van die arteria lienalis vóór operasie gemaak het, en wel op grond van 'n sistoliese geruis oor 'n kloppende geswel wat hy op die röntgenskerm waargeneem het. By outopsie is hierdie diagnose bevestig, maar daar is ook gevind dat die pasiënt aan 'n karsinoom van die pankreas gely het.

In 1921 het Ortner vóór operasie die diagnose gemaak op grond van 'n geswel in die linker bobuik, 'n sistoliese geruis oor die milt, tesame met algemene arteriosklerose. Die diagnose is by operasie bevestig maar die pasiënt is daarna dood.

Die eerste radiologiese diagnose is in 1932 deur Lindboe gemaak op grond van 'n ringvormige verkalking wat agter die bariumgevulde maag geleë was. Ook hierdie diagnose is by operasie bevestig.

In 1952 het Evans deur middel van 'n translumbaal-aortagram 'n pragtige demonstrasie van 'n aneurisma van die arteria lienalis gelewer. Riggs het op hierdie pasiënt met sukses geopereer en die diagnose bevestig.

Owens en Coffey beskou die volgende bevindings as belangrik:

1. Pyn in die linker bobuik,
2. Vergroting van die milt,
3. 'n Tasbare geswel waarin klopping soms klinies waargeneem kan word,
4. 'n Sistoliese geruis oor die geswel,
5. 'n Skaduwee met ringvormige verkalking in die linker bobuik by radiologiese ondersoek, en
6. 'n Vullingsgebrek in die posterior wand van die maag by barium-ondersoek wat soms onder die röntgenskerm duidelike klopping toon, wat volgens hulle diagnosties van die toestand is.

Hulle analiseer die bevindings in 131 gevalle as volg:

Bobuikpyn	71%	Miltvergroting	44%
Mislikheid en braking	30%	Tekens van skok	27%
Geen pyn nie	29%	Buikteerheid	25%
Hematemese	18%	Buikuitsetting	17%
Dispepsie	14%	Röntgentekens	15%
Swakheid	11%	Sistoliese geruis	9%
Melena	11%	Lewervergroting	8%
Kortasem en vertigo	8%	Kloppende geswel	8%
Gewigsverlies	5%	Buikspanning	7%
Neusbloedings	5%	Konvulsies	4%

Owens en Coffey toon verder dat aneurisma van die arteria lienalis meer dikwels in dames voorkom, hoofsaaklik in die swangerskaptydperk, maar met 'n hoogtepunt rondom 48 jaar.

Die volgende faktore is genoem as moontlike etiologiese en patologiese faktore: arteriosklerose (60%), swangerskap (24%), embolië (23%), poortaarhipertensie (20%) aangebore vatafwykinge (10%) en sifilis (6%).

Die moontlike verband tussen poortaarhipertensie en aneurisma van die arteria lienalis word deur sommige skrywers genoem. Die mening bestaan dat enige toestand wat tot verhoogde veneuse druk in die vena lienalis lei, ook mettertyd tot verhoogde druk in die arteria lienalis kan lei, met daaropvolgende ateromateuse veranderings en as gevolg daarvan, aneurisma-vorming.

Afgesien van die gewone oorsaak van poortaarhipertensie naamlik lewersirrose, word ander oorsake van verhoging van veneuse druk in die vena lienalis genoem, soos 'n knak van die vena lienalis met 'n oormatige beweeglike milt, of met buitengewone vergroting van die milt soos met malaria.

Met betrekking tot prognose het Owens en Coffey in hulle analise van 198 gevalle gevind dat, van 124 van hierdie pasiënte wat duidelike simptome as gevolg van aneurisma gehad het, 94 (76%) dood is. Die mortaliteit van die hele reeks d.w.s. waarby ook aneurisma ingesluit is wat geen simptome veroorsaak het nie, was 46%. Hulle wys egter daarop dat alhoewel daar in die verlede so 'n hoë mortaliteit aan die toestand verbonde was, daar in die jongste jare 'n veel meer bemoedigende vooruitsig is. Hoewel die toestand seldsaam is, moet die belangrikheid daarvan nie onderskat word nie.

As die klinikus van die toestand en sy verskynsels bewus is, sal die diagnose beslis meer dikwels vóór

operasie gemaak word. Chirurgiese behandeling sal dan veel meer toegepas kan word met resultate wat oor die algemeen besonder gunstig behoort te wees. Owens en Coffey wys daarop dat die enigste behandeling van die toestand operatief is, en dat operasie aanbeveel behoort te word sodra die diagnose gemaak is, of redelik sterk vermoed word.

Sedert die eerste geslaagde operasie van Winkler is verskeie operatiewe prosedures toegepas. Die operasie waaraan voorkeur gegee word, is verwydering van die milt en van die aneurisma van die arteria lienalis. In hulle reeks is 64 gevalle geopereer, waarvan 30 pasiënte bly lewe het soos hier uiteengesit:

Operasie	*Gevalle*	*Herstel*	*Dood*
1. Miltverwydering alleen	10	6	4
2. Miltverwydering met reseksie of afbinding van die aneurisma	20	15	5
3. Milt verwydering met latere tamponade	1	0	1
4. Afbinding van die arteria lienalis	5	4	1
5. Afbinding en reseksie	6	4	2
6. Tamponade	5	0	5
7. Tamponade en later afbinding	1	1	0
8. Aneurisma afklem	1	0	1
Totaal	49	30	19
9. Nie regstreeks op die aneurisma gerig nie	15	0	15
Groot Totaal	64	30	34

Hoewel voorkeur gegee word aan verwydering van die milt en van die aneurisma, moet die operatiewe prosedure deur ander omstandighede ook beïnvloed word. As die pasiënt erg geskok is as gevolg daarvan dat die aneurisma gebars het, mag dit moontlik betaal om net tydelik die arteria af te bind en verwydering van die aneurisma tot 'n later geleentheid uit te stel. Dit sou egter afhang van die graad van skok en van die verwagte tegniese moeilikhede van die verwydering van die aneurisma in die bepaalde geval.

BESKRYWING VAN ONS GEVAL

J.T., 'n 62-jarige kleurlingvrou, is deur dr. S. Jaffe ingestuur en op 14 Januarie 1954 in die Algemene Hospitaal, Pretoria, opgeneem na 'n aanval hematemese.

Anamnese: Tot ongeveer 2½-jaar vroeër het sy redelik gesond gevoel. Haar eerste simptoom was 'n aanval van kwaai epigastriese pyn wat binne 'n paar minute gevolg is deur floute. Die aanval het oor 'n dag of twee verbeter, sodat sy weer haar werk kon hervat. Na hierdie aanval het sy verskeie dergelike aanvalle van kwaai epigastriese pyn gehad gepaard met 'n skoktoestand. Op minstens twee geleenthede het sy heeltemal haar bewussyn verloor, maar op ander geleenthede het sy net erg pap en moeg gevoel. Met sommige aanvalle het haar dokter haar, weens die kwaai skoktoestand, as 'n noodgeval in die hospitaal laat opneem. Die kliniese beeld was vir hom dié wat gepaard gaan met inwendige bloeding, maar sy het geen verdere uitgesproke tekens van inwendige bloeding getoon nie, en het gewoonlik na twee of drie dae herstel. Met sommige van die aanvalle het sy mislik gevoel en af en toe gebraak.

Vanaf Desember 1952 het sy ook aanvalle van hematemese en melena begin kry. Gewoonlik is die bloeding voorafgegaan deur epigastriese pyn. Sy het in die meeste aanvalle 'n aansienlike hoeveelheid bloed verloor. Sy het gewoonlik met hierdie aanvalle van bloeding nie haar bewussyn verloor nie, maar wel pap gevoel en dors geword.

Tussen aanvalle het sy nog gekla van dowwe pyn in die epigastrium en linker bobuik, en was sy drukgevoelig in hierdie gebiede. Sy kon nie op die linker sy lê nie weens pyn wat dan veroorsaak word. Hierdie epigastriese pyn tussen aanvalle het geen definitiewe verband met ete gehad nie. Sy het heelwat afgeval in die afgelope jaar, maar het haarself nie geweeg nie en kan dus nie presies sê hoeveel sy afgeval het nie.

Daar is geen uitgesproke simptome of afwykings van die hart, longe, urineweë of senuweestelsel nie. Daar is geen vorige geskiedenis van alkohol of hepatitis bekend nie. Geen vorige buikbesering word deur die pasiënt onthou nie. Sy het een kind gehad maar daar was geen moeilikheid by die bevalling nie.

Ondersoek

Behalwe vir matige gewigsverlies, het haar algemene toestand redelik goed voorgekom. Daar was geen opvallende anemie meer nie. Sy het geen geelsug getoon nie. Daar was geen opvallende infeksie in mond, oor, neus of keel nie.

Buik-ondersoek. Met besigtiging was daar geen uitgesette vate op die buikwand nie, en afgesien van 'n volheid wat nie buite normale perke was nie, kon geen verdere afwykinge gesien word nie.

Met betasting was die bobuik altyd effe gespanne sodat dit moeilik was om die onderliggende buik-inhoud te betas. Met versigtige betasting, voordat sy as gevolg van drukgevoeligheid aangespan het, was dit moontlik om 'n geswel in die linker bobuik te voel wat omtrent tot die middellyn gestrek het. Dit het omtrent by die 7de en 8ste ribkraakbene onder die ribrand verdwyn.

Die geswel was omtrent 6 cm. breed in die deel wat onder die ribrand voelbaar was. Dit was moeilik om sy grense te voel, maar dit het die indruk gegee dat dit met asemhaling 'n bietjie afwaarts beweeg. In die gebied van die geswel was daar taamlik duidelike klopping. Dit was egter moeilik om met sekerheid vas te stel of dit net oorgedraagde klopping vanaf die aorta was en of dit uitsettende pulsasie was.

Hierdie geswel is op verskeie geleenthede betas om dit noukeurig te ondersoek maar daar is gevind dat dit soms baie moeiliker tasbaar was as met ander geleenthede. Daar was drukvoeligheid in die linker bobuik en epigastrium. Die indruk was dat hierdie geswel nie 'n vergrote milt was nie, omdat dit te hoog onder die ribrand uitgekom het.

Met beklopping was daar normale resonansie oor die hele buik. Met beluistering kon geen geruise gehoor word nie.

Ondersoek van die res van die buik insluitende rektale ondersoek het geen verdere afwykinge getoon nie.

Ondersoek van die hart het 'n bloeddruk van 140/100 getoon met geen verdere opvallende afwykings nie. Daar is geen kliniese tekens van 'n erge graad van arteriosklerose in die res van die liggaam nie. Ondersoek van die res van die liggaam het ook niks opvallend opgelewer nie.

Spesiale ondersoeke. 18 Januarie, bariummaal-ondersoek met deurligting (T.F.): Hierdie ondersoek is huiwerig op spesiale versoek gedoen, twee dae nadat die pasiënt 'n taamlik kwaai bloeding gehad het, met die gevolg dat sy nie kon staan nie en die ondersoek tot 'n liggende posisie beperk moes word. Sy was baie duiselig. Die doel was om te sien of daar nie miskien 'n maagulkus of karsinoom kon gedemonstreer word om hierdie skerp bloedings te verklaar nie. By deurligting met behulp van 'n bariummaal is 'n hiatusbreuk van die diafragma met terugvloei van barium in die esofagus dadelik waargeneem, maar geen ulkuskrater kon in die esofagus gedemonstreer word nie. Daar was in die linker bobuik 'n vae tasbare geswel net links van die middellyn voelbaar wat hoog onder die ribrand verdwyn het. Die pasiënt was in hierdie gebied erg gevoelig sodat geen doeltreffende betasting op die bariumgevulde maag uitgeoefen kon word nie. Die barium het 'n vae vullingsgebrek aan die posterior wand van die middelgebied van die maag gedemonstreer wat met drukseries sametrekking van die rugae getoon het. Daar was egter geen ulkuskrater in die middel van die sametrekking sigbaar nie, maar die sagte weefselgeswel was duidelik tasbaar in hierdie gebied. Die volgende moontlikhede is tentatief gestel: (1) 'n Bloeiende maagpapilloom met steel aan die posterior-wand, (2) maagkarsinoom en (3) maag-ulkus. (Sien Fig. 1.)

Verdere radiologiese ondersoeke. Op 1 Februarie is 'n presakrale retroperitoneale pneumografie gedoen, en 800 m.l. lug is ingespuit om die niere, byniere en milt beter te omlyn. Die retropneumogram toon duidelik die regter nier en bynier wat soos 'n kappie op die bopool van die nier sit. 'n Digte massa oorvleuel die linker nier en het selfs die vorm van die linker nier. Die milt is duidelik sigbaar onder die linker koepel van die diafragma en is nie vergroot nie. Die retroperitoneale lug het die regter kant beter as die linker kant

Fig. 1. Twee van 'n reeks druk-röntgenfoto's van die barium-gevulde maag in die gebied van die kleinerboog.

gevul. Die linker psoaspier is nie so duidelik sigbaar as die regter psoaspier nie. Geen abnormale verkalking, soos by 'n aneurisma van die arteria lienalis verwag kon word, is waargeneem nie. (Sien Fig. 2.)

Op 2 Februarie is 'n tweede bariummaal met deurvolging gedoen (T.F.): Hierdie keer was die doel van die bariummaal om 'n tentatiewe kliniese diagnose van aneurisma van die arteria lienalis radiologies te probeer bevestig. Die pasiënt was maar baie swak en kon nie bra staan nie, gevolglik was die ondersoek weer hoofsaaklik tot die liggende posisie beperk. Die glyende hiatusbreuk is weereens gedemonstreer maar die esofagus was andersins normaal. Die fundus van die maag het 'n voorkoms in die slymvliespatroon getoon wat sterk verdag was van gastriese varices. By hierdie geleentheid is die sametrekking van die rugae weereens waargeneem maar geen ulkus krater is gesien nie. Dit is dadelik waargeneem dat die geswel wat oor die linker nier duidelik op die retropneumogram gedemonstreer is, nou posisie agter die maag en in die holte van die kleinboog opgeneem het. Die geswel was by hierdie geleentheid baie duidelik tasbaar, maar geen uitsettende pulsasie is waargeneem nie. Daar was ook geen verkalking in die geswel teenwoordig nie. Die geswel was vas aan die posterior wand van die maag en dit verklaar die sametrekking van die rugae. Hierdie geswel was mooi eiervormig en het die kleinboog eweredig uitgedruk en 'n voorkoms getoon wat gewoonlik tipies van 'n supra-gastriese pankreatiese kist is. In die maag self was daar geen bewys van enige patologie behalwe vir die moontlikheid van gastriese varices in die fundus. (Sien Fig. 3.)

Die kwessie om die kliniese diagnose van aneurisma van die arteria lienalis radiologies te ondersteun, kon nie bevestigend beantwoord word nie, inteendeel die röntgenvoorkoms was sterk ten gunste van 'n pankreaskist of karsinoom, alhoewel 'n aneurisma van die arteria lienalis ook nie sover radiologies uitgeskakel kon word nie. Die voorlopige radiologiese diagnose is toe as volg gestel: (1) 'n pankreaskist of karsinoom en (2) 'n aneurisma van die arteria lienalis. Om tussen hierdie twee finaal te besluit is 'n translumbaal aortografie aanbeveel.

Ondersoeke by 'n vorige opname. November 1953

(1) *Bariummaal.* 'n Glyende hiatus-breuk met esofageale varices is waargeneem. Geen verdere patologie is in die spysverteringskanaal gedemonstreer nie.

(2) *Esofagoskopie* het by een geleentheid moontlike esofageale varices getoon, terwyl 'n herhaling by 'n ander geleentheid geen uitgesette venae gedemonstreer het nie.

(3) *Maagsapanalise* het geen vry soutsuur getoon nie en die totale suur was 8 eenhede.

(4) *Lewerfunksie.* Die proewe het die volgende getoon: Totale proteïene 5·85 g.% (albumine 2·84 g.%, globuline 3·01 g.%); Timol-troebeling 0; timol-vlokking 0; kolloïde goud 0; totale bilirubin en van der Berg—kleurloos. Lewerfunksie dus normaal.

(5) *Bloedondersoek.* Hemoglobin 9·4 g.% (63.5%); rooiselle 3,540,000; witselle 5,500, met normale differensiële telling. Kolmer- en Price-toetse was negatief.

(6) *Diagnose.* Die pasiënt is destyds na herstel van die bloeding met 'n diagnose van 'n glyende hiatusbreuk met esofageale varices ontslaan.

DIE TEENSWOORDIGE DIAGNOSE

Die diagnostiese probleem by hierdie geleentheid was dus dié van 'n 62-jarige kleurlingvrou wat die volgende getoon het:

1. Aanvalle van epigastriese pyn wat vroeër in die siekte deur floutes gevolg is en later deur hematemese en melena.

2. 'n Tasbare geswel in die linker bobuik wat egter nie met elke geleentheid gevoel kon word nie. Hierdie geswel het geklop maar dit was onseker of dit oorgedraagde of uitsettende pulsasie was.

3. Drukgevoeligheid in die linker hipochondrium en epigastrium.

4. 'n Esofageale hiatusbreuk van die glyende tipe.

5. Radiologiese bewys van 'n ronde geswel agter die maag en vas aan die posterior-wand, met moontlike gastriese varices, maar geen verkalking in die geswel nie.

6. Geen radiologiese tekens van patologie in die maag self nie soos maag-ulkus, maagkarsinoom of papilloom nie.

Die volgende toestande moes toe as oorsaak oorweeg word:

1. 'n Geswel wat sy oorsprong in die pankreas gehad het soos bv. 'n pankreaskist. Hiermee sou die geswel verklaar kon word, maar die pankreaskist alleen sou nie die bloedings kon verklaar nie. Dit was egter moontlik dat so 'n geswel in die pankreas tot druk op die vena lienalis aanleiding kon gegee het (moontlik met trombose

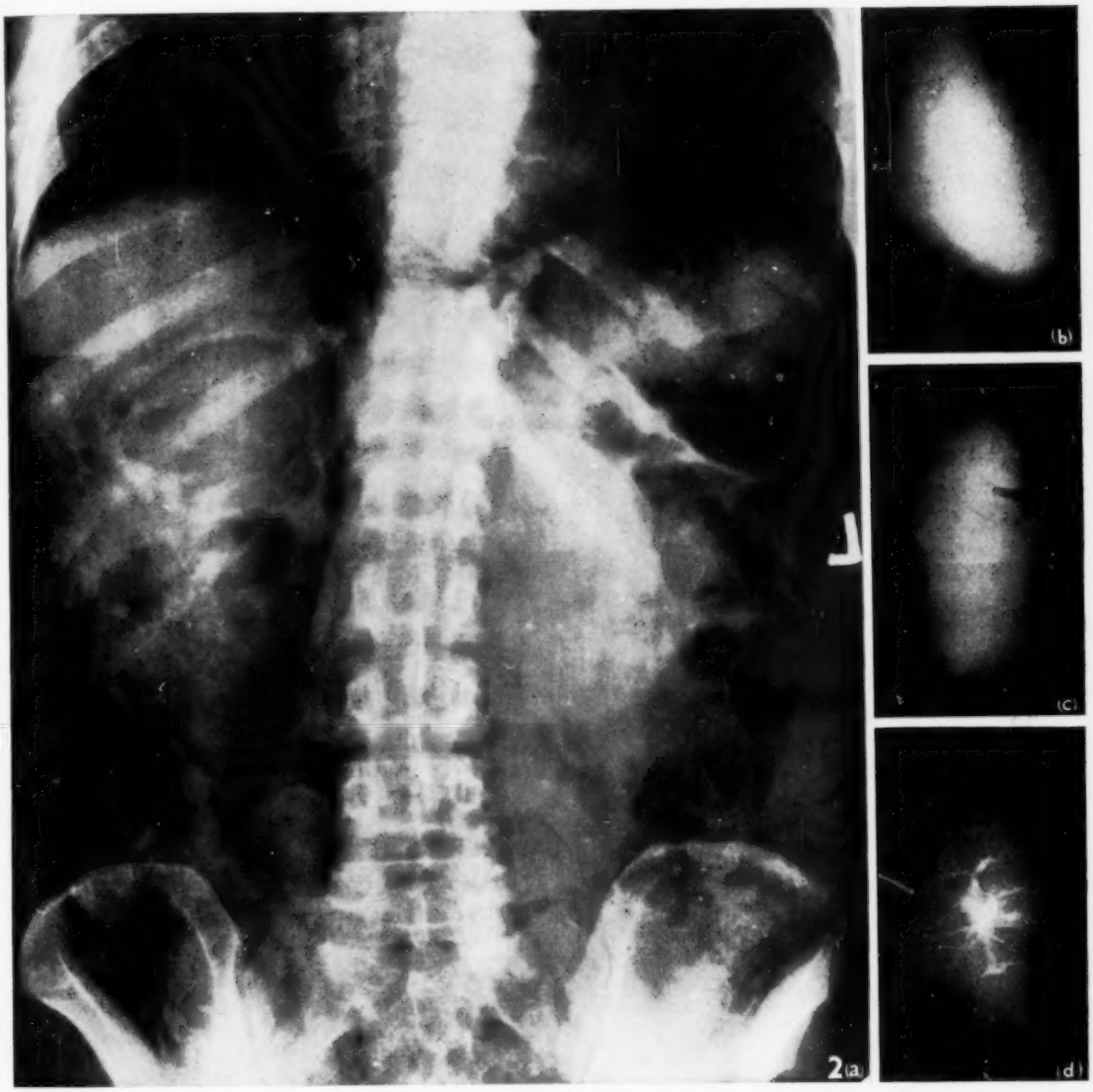

Fig. 2. (*a*) 'n Retropneumogram wat die eiervormige geswel oor die linker nier in die linker bobuik demonstreer. (*b*) 'n Röntgenfoto van die verwyderde milt. (*c*) 'n Röntgenfoto van die verwyderde aneurisma van die arteria lienalis gevul met gestolde bloed. (*d*) 'n Röntgenfoto van die vate van die milt met radio-ondeurskynende kleurstof daarin gespuit. Die vate is normaal in voorkoms.

daarvan) met daaropvolgende stuwing in die gebied van die vena lienalis en sy takke insluitend die vasa brevia vanwaar die bloeding kon kom.

2. Aneurisma van die arteria lienalis kon ook redelik goed hierby inpas, want dit sou die kloppende geswel kon verklaar en dit sou die bloeding kon verklaar, deurdat daar herhaaldelik lekkasie vanaf die aneurisma direk in die maag plaasgevind het, of op andere manier wat in die bespreking genoem sal word. Die onsekerheid van die aard van die pulsasies wat klinies voelbaar was en die seldsaamheid van die toestand het hierdie moontlike diagnose bietjie onseker gemaak.

3. Indien die diagnostiese moeilikheid alleen die van hematemese en melena sou gewees het, was daar verdere moontlikhede soos peptiese ulkus, diafragmabreuk en esofagus-ulkus, poortaarhipertensie. Hierdie toestand sou egter nie die bevinding van 'n geswel in die linker bobuik verklaar nie en kon dus buite rekening gelaat word, behalwe indien die moontlikheid van dubbele patologie oorweeg moes word.

4. Die radiologiese beeld het maagkarsinoom uitgeskakel.

'n Verdere ondersoek wat deur die radioloog aanbeveel is en wat seker 'n finale antwoord kon gegee het was

Fig. 3. 'n Tweede bariummaal na retropneumografie. Die geswel is agter die maag en oefen eweredige druk uit op die holte van die kleinboog. Die fundus van die maag toon die verdagte voorkoms van esofageaal-varices.

translumbaal-aortografie. Terwyl dit egter duidelik geword het dat die operasie in elk geval nodig sou wees, en vanweë die pasiënt se swak toestand, is besluit dat hierdie ondersoek onder die omstandighede nie geregverdig was nie, en daarom is dit toe nie gedoen nie.

Daar is tot operasie besluit met die waarskynlike diagnose van 'n aneurisma van die arteria lienalis met 'n alternatiewe diagnose van 'n geswel in die gebied van die pankreas.

OPERASIE

Operasie is op 11 Februarie in die nie-blanke Afdeling van die Algemene Hospitaal, Pretoria deur een van ons (J.K.B.) gedoen. Algemene narkose is deur dr. Pen Wessels toegedien en dr. P. Hack en dr. D. Botha het by die operasie assisteer.

Die buik is deur 'n lang, linker paramediaan-snit oopgemaak. 'n Kloppende geswel kon duidelik agter die maag gevoel word. Dit was van kant tot kant taamlik vry beweeglik en tot 'n mindere mate ook van bo na onder. (Hierdie beweeglikheid verklaar waarom die geswel nie altyd ewe duidelik voelbaar was nie.) Aan die onderkant van die mesokolon transversum kon duidelik gesien word dat die kloppende geswel aan die mesokolon vasgekleef was. Voordat die geswel verder blootgelê is, is roetine-ondersoek van die res van die buik gedoen. Die lewer het normaal voorgekom. Die milt was binne normale perke. Die venae langs die groot en klein boë van die maag het nie erg uitgeset voorgekom nie, behalwe vir een of twee vasa brevia wat ietwat dikker as normaal gelyk het. Geen maag-ulkus of ander afwyking in maag of duodenum is gevind nie. In die res van die buik was geen opvallende afwyking nie.

Die kleinsak is deur die omentum majus oopgemaak sodat 'n redelike groot opening geskep is. Hierdeur kon die aneurisma

gesien word. Dit het van die gebied van die arteria lienalis ontspring (waarskynlik 2 tot 3 cm. van sy oorsprong van die arteria coeliaca) en het afwaarts en na vore gestrek sodat dit die liggaam van die pankreas afwaarts gedruk het en laer af aan die mesokolon verkleef het. Op sy anterior oppervlak was die aneurisma aan die posterior-maagwand so vasgekleef dat dit later met die mes losgesny moes word. (Dit verklaar die sametrekking van die rugae van die maag wat by bariumdeurligting waargeneem is.)

Die grootte van die aneurisma was ongeveer 10×5 cm. en die vorm min of meer eiervormig. Die proksimale deel, d.w.s. die deel naaste aan die arteria self, het geklop, maar die distale deel het betreklik min klopping getoon a.g.v. stolling van die bloed daarin. Een deel, wat tot die mesokolon transversum gestrek het, was met 'n sandlopervernouing van die res afgesonder en het gladnie geklop nie.

Die arteria lienalis proksimaal tot die aneurisma is gevind en deurklief. Die distale deel van die arteria lienalis het betreklik dun gelyk en is toe afgebind. In hierdie stadium het dit nog gelyk asof dit 'n bietjie moeilik sou wees om om die aneurisma te kom en daarom is besluit om die milt los te maak met die bedoeling dat dit saam met die stert van die pankreas na vore getrek kon word.

Die losmaak van die milt het egter nie veel gehelp nie want die pankreasstert is afwaarts weggedruk en het nie mooi saam met die milt na vorentoe gekom nie. Die milt is verwyder.

'n Taamlike uitgesette vena wat net bokant die aneurisma gelê het is aan albei kante onderbind en die stuk saam met die aneurisma verwyder. Dit het in die rigting van die miltsteel geloop en was moontlik die vena lienalis of 'n groot tak daarvan, hoewel die ligging bokant die arteria nie die gewone ligging is nie.

Die proksimale deel van die aneurisma is uitdissekteer maar die distale deel waar dit in die mesokolon inloop is oopgemaak, die stolsel uitgehaal. en die oppervlakte met peritoneum bedek. Die hele soliede deel in die masokolon is met rus gelaat.

Aan die diafragmabreuk is niks gedoen nie. 'n Dreineringsbuis is in die kleinsakgebied ingebring. Die wond is op die gewone manier gesluit. Sy het na operasie goed herstel.

Vyf maande later is dit berig dat die pasiënt nog goed voel en geen verdere moeilikheid gehad het nie. Ongelukkig het ons nog nie weer die geleentheid gehad om haar in hierdie stadium te ondersoek nie.

BESPREKING

Wat die etiologie van die aneurisma in hierdie geval betref moet arteriosklerose as waarskynlikste oorsaak beskou word. Die Kolmer-reaksie was negatief en daar was geen geskiedenis van besering nie. Ten spyte van die afwesigheid van besondere opvallende tekens van arteriosklerose moet dit op haar ouderdom tog as die waarskynlikste oorsaak beskou word.

In verband met die kliniese beeld en die diagnose sal hier geen volledige bespreking gegee word nie daar die artikel van Owens en Coffey, waarna alreeds verwys is, 'n baie breedvoerige bespreking van hierdie aspekte gee. Die bespreking sal dus tot enkele punte beperk word.

Die verklaring van die aanvalle van epigastriese pyn wat vroeër in die verloop van die siekte deur floute gevolg is, is waarskynlik aan die feit te danke dat die aneurisma op hierdie geleentheid gelek het maar nie in die spysverteringskanaal deurgebreek het nie. Later in die verloop van die siekte het dit wel in die spysverteringskanaal deurgebreek en die waarskynlikste plek volgens die operatiewe bevindings is 'n direkte lekkasie deur die posterior wand van die maag.

Hoewel in hierdie geval die bloeding onstaan het a.g.v. 'n direkte deurbraak van die aneurisma in die maag, kan bloeding in pasiënte met aneurisma van die arteria lienalis moontlik ook op ander maniere ontstaan soos hieronder uiteengesit:

1. Eerstens is daar die moontlike verband met poortaarhipertensie. Daar is skrywers wat meen dat in pasiënte met poortaarhipertensie daar mettertyd nie alleen in die vena lienalis maar ook in die arteria lienalis 'n verhoogde druk ontstaan en dat hierdie verhoogde druk tot ateromateuse veranderinge in die arteria lienalis, soms met daaropvolgende aneurisma, aanleiding gee.

2. 'n Verdere moontlikheid in verband met hipertensie in die lienalis-deel van die poortaarstelsel is dat die aneurisma moontlik op die vena lienalis soveel druk mag uitoefen dat dit afgesluit word of trombose ondergaan, en as gevolg hiervan daar 'n toename in die veneuse druk in die vena lienalis is met daaropvolgende bloeding van die maag. Hierdie moontlikheid skyn nie voorheen genoem te wees nie maar dit lyk tog, op teoretiese grond, of dit sou kon gebeur. Dit is moontlik dat in gevalle van aneurisma van die arteria lienalis wat nie 'n ander oorsaak vir poortaarhipertensie toon nie, miltvergroting ten gunste van hierdie teorie sou spreek.

3. 'n Derde moontlikheid as oorsaak van bloeding in die spysverteringskanaal in gevalle van aneurisma van die arteria lienalis, is aan een van ons (J.K.B.) getoon tydens 'n onlangse reis oorsee. In Utrecht, Holland, het prof. J. F. Nuboer 'n pasiënt geopereer op wie hy die diagnose van lienalis-aneurisma op radiologiese gronde gemaak het. Hierdie pasiënt was 'n man in sy 50ste jaar wat aan herhaalde aanvalle van melena gely het, en met ondersoek uitgesproke tekens van arteriosklerose elders in die liggaam getoon het. By operasie is 'n aneurisma van die arteria lienalis gevind wat 'n groot deel van die pankreas vernietig het en uiteindelik in die hoofpankreasbuis gebars het, sodat daar bloeding vanaf die aneurisma via die pankreasbuis in die spysverteringkanaal plaasgevind het. Met die toestemming van prof. Nuboer maak ons melding van hierdie seldsame manier waarop bloeding vanaf 'n lienalis aneurisma die spysverteringskanaal kan bereik.

Wat die radiologiese ondersoek betref, het die bariummaal-deurligting gepaard met retropneumografie 'n ronde of liewer ovale geswel wat agter die maag geleë en aan die posterior wand van die maag vasgekleef was, gedemonstreer. Die retropneumografie het baie gehelp om die geswel met betrekking tot die niere en milt duideliker op te wys, en dit was gevoel dat 'n translumbaal-aortografie die finale diagnose sou beklink het.

Wat die behandeling betref skyn dit wenslik te wees om indien moontlik die aneurisma te verwyder. In hierdie geval was die verwydering van die milt waarskynlik nie nodig nie, maar dikwels is dit wenslik.

OPSOMMING

'n Beskrywing word gegee van 'n pasiënt met 'n aneurisma van die arteria lienalis wat voor operasie tentatief gediagnoseer is op grond van pyn in die linker bobuik, hematemese en melena, kloppende geswel in die linker bobuik en die radiologiese aanduiding van 'n ovale geswel agter die maag in die gebied van die arteria lienalis. Die diagnose is operatief bevestig en die aneurisma van die arteria lienalis is met sukses verwyder. Die pasiënt verkeer, volgens mededeling, nou 5 maande na operasie in goeie gesondheid.

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TOPICAL NEOMYCIN IN CERVICAL AND VAGINAL INFECTIONS WITH SPECIAL REFERENCE TO BACILLUS PROTEUS INFECTIONS

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Antibiotics has been a boon in the treatment of cervical and vaginal infections, especially in their relation to sterility. Even chronic cases have reacted surprisingly well to antibiotic therapy and the question of topical applications directly to the cervix and intracervically has been discussed by the author before.[1, 2] Aureomycin, crysticillin, streptomycin and sulphonamides have been used for this purpose in the form of ointments or solutions. The oral and parenteral use of antibiotics guided by sensitivity tests yielded good results, but their topical use has not been very successful, especially in chronic cases and in particular in bacillus proteus infections.

It is a well-known fact that B. proteus in the cervix and vagina is extremely resistant to treatment. In bladder infections intramuscular streptomycin has proved successful, but in cervical and vaginal lesions neither streptomycin nor other antibiotics or chemotherapeutic agents have been of any distinct value. Frequently proteus is not found in the original vaginal and cervical culture, but appears only after the disappearance of the primary bacillary infection, particularly haemolytic streptococci and staphylococci. Seeing that many of the oral antibiotics cause a slight diarrhoea, may it be possible that they predispose to the appearance of the proteus, or does this scavenger appear only after the primary pathogens of cervix and vagina have been destroyed?

The spermicidal tendencies of *Escherichia coli*, *Streptococcus viridans*, haemolytic streptococci, staphylococci and *Proteus vulgaris* are well known.[2, 3] I advocated [2] the combination of oral or parenteral antibiotic treatment with local antibiotic intracervical and vaginal therapy in order to obtain the best results in cases of infection and found it therefore of interest to investigate the efficacy of neomycin-sulfate cream (Myciguent Cream), which was kindly put at my disposal.

TABLE I

Number of cases	*Bacterial infection*	*Anti biotic used*	*1st bacterial flora after treatment*	*2nd bacterial flora after treatment*	*Pregnant after treatment*
1	StaAuH BD, BP	T.	nil	—	yes
2	BC, BP	T.	nil	—	yes
3	StaAuH StaNH BD.	T.	nil	—	—
4	StaAuH StrV.	T	M StrNH StrV BP	nil	yes
5	StaAuH BD	T.	StrV. StaAlb BP	nil	—
6	StrH BP	T.	StrV BP	nil	yes

TABLE I (*continued*)

Number of cases	*Bacterial infection*	*Anti biotic used*	*1st bacterial flora after treatment*	*2nd bacterial flora after treatment*	*Pregnant after treatment*
7	EC, BP	SM	StrV BP	nil	yes
8	BP	SM	nil	—	—
9	BGNH StrNH	C	nil	—	—
10	EC, BD StaAuH BP	T	nil	—	—
11	EC, BP	T	StaAuH T	nil	yes
12	StrH StaAuH BD	T	nil	—	yes
13	BP	SM	nil	—	—
14	L, StrH	T	nil	—	—
15	StaAuH BD	C	M	M	—
16	BP StaAuNH	T	BP	nil	—
17	StrH	C	nil	—	—
18	StaAuH T	T	T	nil	—
19	StaAuH StrV, BD StrH	T	BP Str	nil	—
20	StrNH StaAuH BD, T	E	BP, T	nil	—
21	T, BP StaAlb	T	BP	nil	yes
22	StaH, BD	T	nil	—	—
23	StrH StrNH	T	BP	nil	yes
24	StaAuH	T	EC, BP	nil	—
25	StrH, BC StaAuH	SM	StaAuH BP	nil	—
26	StaAu	T	StaAuNH	StaAuNH	—
27	StaAu BD, M	T	M	M	—
28	BC, M	T	M	nil	—
29	StaAuH BD	T	M	nil	—
30	StrH, BD	T	nil	—	yes
31	StrNH StaNH	C	nil	—	—
32	StaAuH BD	T	StaAuH StaAuNH	nil	—
33	StaAuH	T	StrNH BP	nil	—
34	StrH	T	nil	—	yes
35	StaAu T	E	nil	—	yes
36	StrV BD, BP	T	BP	nil	yes
37	StaAuH BD	T	M	nil	—
38	StaAu StrNH BD	C	nil	—	—
39	StaAuH BP	T	BP	nil	yes
40	EC, BP	SM	BP	BP	—

TABLE I (*continued*)

Number of cases	*Bacterial infection*	*Anti biotic used*	*1st bacterial flora after treatment*	*2nd bacterial flora after treatment*	*Pregnant after treatment*
41	StaAu BD	T	nil	—	yes
42	StrV StaAuH BD	T	BP	nil	—
43	StaAu, M	T	nil	—	—
44	StaAuH StaAuNH	T	BP	nil	—
45	StrV StrH	T	nil	—	yes
46	BP StaAuH	T	nil	—	—
47	StaAuH StaNH, BD	T	StrH StaAlb BP	nil	—
48	BGN	E	nil	—	—
49	EC, BP	SM	BP	nil	—
50	StaAuNH BD	C	BP	nil	—
51	T, StrH	T	M	M	—
52	T, StrH StaAlb BD	T	T, BP	T	—
53	StaAuH BD StaAuNH	T	nil	—	yes
54	StrH, StaAuH StaAuNH	C	M	M	yes
55	EC, BP	SM	StaAuH M	nil	—
56	StaAuH BD, BP	T	BP	nil	yes
57	StrNH StrV, BD	T	nil	—	yes
58	StaAu T, BD	T	BP	nil	yes
59	StrV, T, BD	T	BP	BP	—
60	StaAu BD	T	nil	—	yes
61	EC, BD StaAu	T	BP	nil	yes
62	StrV, BD StrNH	C	nil	—	yes
63	StaAu, BP	T	BP	nil	yes
64	StrH StaAu	T	nil	—	—
65	StaAu, T, BP	T	BP	nil	yes
66	StrNH BD	T	nil	—	yes
67	StaAuH BP, BD	T	nil	—	yes
68	T, StaAuH	T	BP	nil	yes
69	StaAlb, T	T	nil	—	yes
70	StaAu, BP	T	nil	—	yes

Bacteria:

BC .. Coliform bacillus.
BD .. Diphtheroid bacillus.
BGN .. Gram-negative bacillus.
BGNH .. Haemolytic gram-negative bacillus.
BP .. Bacillus proteus.
EC .. Escherichia coli.
L .. Leptothrix.
M .. Monilia.
StaAlb .. Staphylococcus albus.
StaAu .. Staphylococcus aureus.
StaAuH .. Haemolytic Staphylococcus aureus.
StaAuNH Non-haemolytic Staphylococcus aureus.
StaH .. Haemolytic Staphylococcus.
StaNH .. Non-haemolytic Staphylococcus.
StrH .. Haemolytic Streptococcus.
StrNH .. Non-haemolytic Streptococcus.
StrV .. Streptococcus viridans.
T .. .. Trichomonas.

Drugs:

C Crysticillin. SM Streptomycin.
E Erythromycin. T Terramycin.

Table I tabulates results in 70 cases of cervical and vaginal infection. In all cases oral or parenteral antibiotic treatment has been combined with repeated topical intracervical and vaginal application of neomycin-sulfate cream. The general and topical use of antibiotics in the treatment of cervical and vaginal infection had considerably improved the likelihood of cure, but the topical use of neomycin combined with oral or parenteral antibiotics has further increased the percentage of cure.

In a previous communication [2] (prior to the introduction of topical neomycin) I was able to report that in 34 cases of cervical and vaginal infection pregnancy resulted in 12 cases (34%) after treatment by the general and topical use of antibiotics. After the combined use of antibiotics and topical neomycin pregnancy resulted in 31 out of 70 cases treated (i.e. 44.5%).

This is a considerable improvement, especially as cases of very long standing responded surprisingly well. I would like to quote case 4:

> Mrs. H.S. was trying to have an infant for 9 years and was suffering from cervical and vaginal bacterial infection with haemolytic *Staphylococcus aureus* and *Streptococcus viridans*. Oral treatment with 48 capsules Terramycin mgm 250 over 8 days and daily intracervical and vaginal topical application of neomycin cream was instituted. The second bacteriological examination carried out 8 days after therapy was commenced showed monilia, non-haemolytic streptococcus, *Streptococcus viridans* and *Proteus vulgaris*. The patient was then treated daily for 10 days with topical application of neomycin alone; the third bacteriological examination showed no pathological organisms and within 3 weeks from the beginning of treatment the patient fell pregnant.

In most cases the second bacteriological examination was done within 8 days of the institution of therapy and if the necessity arose a third one within 3 weeks from the beginning of treatment.

No side-effects have been observed from the use of topical neomycin. Neomycin did not affect trichomonads or monilia.

The most interesting finding was the almost specific effect on *Proteus vulgaris*. The incidence of this organism and the effects of treatment are shown in Tables II and III.

TABLE II. INCIDENCE OF BACILLUS PROTEUS

Bacillus proteus present	*Cases*
In mixed bacterial infections before treatment ..	21
In the first bacterial flora after treatment ..	28
For the first time in the bacterial flora after treatment	17
Persisting in the second bacterial flora after treatment	2
Total	38

TABLE III. RESULTS OF TREATMENT

Bacillus proteus present	*Cases*
Responding to initial treatment with antibiotics and topical neomycin	10
Responding to topical neomycin application alone	26
Resistant to topical neomycin	2
Total	38

Thus the proteus infection was eradicated in 36 out of 38 cases.

Of the 38 cases of proteus infection treated with antibiotics and topical neomycin 18 resulted in pregnancy.

I attribute these excellent results to the application of topical neomycin and I recommend its use in proteus infections.

SUMMARY

1. Seventy cases of cervical and vaginal infection, of which 31 resulted in pregnancy, have been presented.
2. All these cases have been treated with the appropriate antibiotics in combination with topical neomycin.
3. Amongst these 70 cases there were 38 cases of infection with *Proteus vulgaris*.
4. Ten cases of proteus infection responded to initial treatment with antibiotics and topical neomycin and 26 to the use of topical neomycin alone; i.e. over 90% of cures.
5. Of 38 cases of proteus infection 18 cases resulted in pregnancy after treatment.
6. The use of topical neomycin alone or in combination with other antibiotics is of distinct value in cervical and vaginal infection, especially when bacillus proteus is present.

I am grateful to Dr. Harold R. Reames, Ph.D., M.D. Medical Division Department of Clinical Investigations, The Upjohn Company, Kalamazoo, Michigan, who supplied the neomycin-sulfate cream (Myciguent Cream).

My special thanks are furthermore due to Dr. W. Lewin, Pathologist, Johannesburg, for the bacteriological work.

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REVISION SERIES

V. THE MANAGEMENT OF MALARIA

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Too much emphasis has perhaps been placed on what constitutes the dosage of an anti-malarial drug, since most assessments refer to cases of average severity. Any such estimation, especially in a disease like malaria, tends to overlook the clinical condition of the patient. One cannot predict how much of the selected drug is required or how long the course will last. The principle applies to any state of fever but in no disease perhaps is this point of greater significance than in malaria, for failure to appreciate it may mean the loss of a life.

THE SEVERE GROUP

Before deciding on the dosage and method of administration of an antimalarial drug, one must assess the clinical state of the patient. Signs of severity are indicated by:

1. Restlessness.
2. High fever for 2 or more days.
3. Vomiting (repeated).
4. Severe and persistent headache.
5. Although a rough guide, the more parasites *per* field as seen microscopically in the peripheral blood, the more seriously ill is the patient.

These features are more likely to be met in the non-immune subject exposed for the first time, although this rule is not absolute; in an immune subject a heavy infection occasionally occurs and the patient may be dangerously ill. Generally speaking, any of the following may be regarded as belonging to the serious or potentially serious group:

1. Acute fever in an infant or child.
2. Acute fever associated with dysentery or severe diarrhoea.
3. Acute fever with jaundice (bilious remittent fever).
4. Signs of collapse with a weak pulse, a fever which is moderate or only slight, and signs of renal impairment (protein and casts in the urine and a raised blood urea).
5. Acute fever in a person beyond middle age infected for the first time.
6. An untreated case of acute fever of a few or several days' duration.

For the severe group quinine is the drug which should be employed unless its use is contra-indicated; for it has a rapid and specific action with a marked effect against asexual erythrocytic parasites. When a subject with malaria is acutely ill, quinine is given at first by intravenous injection but subsequent doses are administered according to the condition of the patient. This must depend on the practitioner's own judgment. If there is much vomiting, signs of collapse, loss of fluid or delirium, an intravenous drip infusion (glucose-saline) is set up at once and quinine dihydrochloride (gr. 10 for an adult, half this amount for a child and about gr. 4 for an infant) is injected into the rubber connection (not into the bottle), the drip flowing in at a rate of 30 drops per minute. This method ensures a slow, steady and safe administration of the drug. The quinine is repeated in the same way every 6 hours until the

patient improves, when it can be given intramuscularly. The drip infusion is continued for 24—48 hours or even longer when necessary, the saline being alternated with 5% glucose. If it is not possible to set up a drip infusion the quinine is slowly injected intravenously in 10 c.c. of sterile normal saline and this is repeated after 6 hours if necessary. It must be injected slowly at a rate not exceeding 1 gr. per minute. Should the fever fail to respond within 24—36 hours, adrenalin hydrochloride, m. 8 every 6 hours subcutaneously, appears to be of value in assisting to lower the temperature.

Where the patient is acutely ill but his general condition is satisfactory, the quinine is best given by the intramuscular route. For an adult the dose is 10—15 grs. of quinine dihydrochloride to be repeated in doses of 7½—10 grs. every 6 hours. But if after the 1st or 2nd injection the patient has sufficiently recovered the quinine may be taken by mouth. For older children the dose is 5—7½ grs. and for an infant 3—5 grs.

When quinine is exhibited intramuscularly great care must be taken to ensure that it is injected into the correct anatomical site and that the equipment employed is carefully sterilized. The doctor should administer the injection himself and not leave this responsibility to the nurse. The site should be carefully mapped out in the upper and outer quadrant of either buttock. Each injection is given in an alternate buttock and, when a second injection is to be given in the same buttock, the needle must be inserted at least 1½ cm. distant from the site of the previous one. The needle must be inserted deep into the muscle and not under the skin, as frequently occurs. If bone is contacted the needle need only be withdrawn ½ cm. away. As to size, the needle should be a Record No. 19 and 2 inches in length. It is best to have 2 needles and a dry syringe. The physician washes his hands well and draws up the quinine into the syringe fitted with one of the needles. After the skin area has been sterilized with an antiseptic the second needle is plunged into the muscle, the syringe is attached to it and the injection made.

When the patient is over the acute phase the quinine may be continued by mouth, gr. 10 2 or 3 times a day for 7—10 days. Chloroquine, 1 tab. b.d. for 6 days, or a combination of quinine, gr. 5 t.i.d., with chloroquine, 1 tab. b.d. for 5 days, may be substituted.

I have purposely avoided much reference to mepacrine for acute fever, because it has one serious disadvantage, namely the risk of mepacrine psychosis. Although the risk is slight I have encountered several cases following its administration in relatively small amounts. It has however a limited use when quinine is contra-indicated. One example is fever in pregnancy, where mepacrine can be relied upon to produce a fairly quick response. Like camoquin, its principal action is against asexual erythrocytic parasites, and it resembles quinine in being ineffective against the gametocytes of *P. falciparum*. Mepacrine hydrochloride is best administered intramuscularly for the severe group, in doses of 300 mg. or less for an adult, depending on the response, repeated 6-hourly.

Cerebral Malaria. As cerebral malaria is such an important complication of malignant tertian malaria it is justifiable to mention it separately although the principles for this serious form of malaria are the same as those already mentioned for the 'severe' group of cases. The treatment is much the same except that in cerebral malaria quinine must be given by the intravenous route after a glucose-saline drip infusion has whenever possible, been set up. This applies to all ages. Intramuscular quinine alone is not enough, and reliance must be placed on intravenous quinine and fluid. The amount of quinine varies very greatly from case to case, but for an adult 10—15 grs. of quinine dihydrochloride are normally given every 6 hours until signs of recovery set in, the quinine being injected into the rubber connection. For children the dose is 5—7½ gr. every 6 hours and for infants 4—5 gr. at similar intervals. Antibiotics are of no special value unless later in the course of the illness. If congestion of the lungs supervenes it may be given as a preventive against pneumonia. When quinine is contra-indicated, mepacrine may be employed and can be given intravenously, the dose being 300 mg. injected into the tubing of the drip saline.

THE ORDINARY ACUTE ATTACK OF MALARIA

For this group the selection of the particular anti-malarial is not of such importance as in the more acute and serious cases previously described, although it should not be dismissed lightly. There are a number of extremely valuable drugs available and the prospects of cure are extremely good.

Where possible I prefer not to give quinine, because it is not altogether without risk. Especially in the patient who has had repeated acute attacks of malignant tertian malaria, it may precipitate an attack of blackwater fever. But it must be pointed out that haemoglobinuria may follow any of the anti-malarial drugs. Quinine is of risk to a pregnant woman and when larger doses are given may induce a miscarriage. In the sensitive it may also produce unpleasant effects on the hearing, which in some cases are permanent, and occasionally it causes amblypopia. All these risks are admittedly slight, but when one anticipates a complete recovery with reasonable certainty of eradication of the parasite from the body no risk is justifiable if a safer drug is available. In my experience chloroquine is safe and the side-effects are comparatively slight.

For this group therefore, I would recommend chloroquine, which may be obtained as chloroquine diphosphate (aralen) each tablet of 250 mg. containing 150 mg. of base, or chloroquine sulphate (nivaquine B) a tablet of 200 mg. containing 150 mg. of base. I commence with 4 tablets of aralen or nivaquine B at once, followed in 6 hours by 2 tablets, and after that 1 tab. twice daily for 3 days. Also effective is camoquin (amodiaquin dihydrochloride dihydrate) (each tablet containing 200 mg.) of which the dosage is 3 tablets on the first day as a single dose and followed by 2 tablets daily for a further 2 days. Should the clinician for various reasons prefer to treat with quinine or mepacrine the following course is recommended:

Mepacrine hydrochloride 200 mg. t.i.d. on the 1st day, 200 mg. on the 2nd day and thereafter 100 mg. t.i.d. for 7 days. For quinine sulphate or dihydrochloride 10 gr. t.i.d. for 7—10 days.

For the acute attack in this group it is good treatment before administering the antimalarial drug first to induce sweating with aspirin or a Dover's powder and placing the patient between blankets. This will aid in lowering the fever, after which camoquin, chloroquine or quinine may be given—usually within an hour.

Relapses with acute fevers are as a rule not as severe as the first attack and, further, the patient usually responds more quickly to antimalarial drugs. Once again the same drugs are recommended, but in somewhat smaller dosage and the course may be reduced to about 5—7 days, the patient being allowed out of bed shortly after the fever has returned to normal. Relapses in malignant tertian malaria occur, but not as often as in the vivax or quartan varieties of the disease, which are far less common in Africa except in certain areas. I have at times employed plasmoquin for relapse vivax malaria but have found it disappointing and as plasmoquin may be accompanied by unpleasant reactions, some of which are severe, I would prefer not to recommend its use. As a rule the disease clears up satisfactorily, but in the meantime I prefer to rely on a course of the safer antimalarials combined with a short course of arsenic (N.A.B., O.3 g.—once a week for about 5 or 6 weeks), a ferrous sulphate mixture and vitamin therapy, ensuring that the patient takes the necessary precautions against reinfection.

It is worth while referring to the acute relapse of fever that may follow an operation or delivery, for it is not uncommonly encountered in practice. Unless one is on the alert and remembers this fact the cause of the fever may be overlooked. It is therefore a golden rule, when caring for a patient who has undergone a surgical procedure or has just given birth to a child, to remember this possibility should the temperature rise. Such a relapse may be mild, but occasionally it is serious. The dosage will depend on the severity of the malarial infection.

The management of malaria in the European and Bantu varies greatly, largely because of environmental factors and because the latter in endemic regions has a strong immunity acquired over many years. The European therefore, on the whole, requires a larger and longer course of treatment. The immune Native, especially when returning to his same environment, requires as a rule considerably less. In any event, many do not require hospitalization and refuse to seek medical advice, the fever subsiding spontaneously in a day or two. The average case requires quinine, gr. 10 t.i.d. for about 2—4 days, or chloroquine, 1 tab. b.d. for 2 days, and the milder cases respond well to aspirin gr. 10. It is important to remember that the Native living in an endemic area may develop an acute illness like pneumonia, typhoid, meningitis, whilst at the same time the peripheral blood may reveal the presence of malarial parasites. This is because so many of the Bantu harbour parasites in the blood. Fortunately this is not likely to be mistaken for malaria alone if the case is examined carefully. It is far more important to treat the non-malarial infection, although antimalarial therapy for the malarial parasites may or may not be given, depending on the circumstances of the case. This picture of dual acute pathology is less liable to be met with in the European patient. In the European, therefore, if malarial parasites are found in the blood one may presume the patient is suffering from malaria and the treatment given must be vigorous and intense. In a Native on the other hand, even though the parasites be present in the blood film, care should be taken to make sure that the patient is not suffering from a more serious infection elsewhere in the body, particularly one which is not immediately obvious.

If from the clinical history and examination a case appears to have malaria, and yet the smears are negative, the patient should be treated energetically with the selected drugs in accordance with the clinical picture. This applies more so to the European and non-immune Native, in whom treatment must be prompt and adequate. In the immune Native, when one is in doubt one can afford to delay the institution of treatment for a short time.

Chronic Malaria. The diagnosis of this form of malaria is extremely difficult and is less often made today than in former times. It should be made only in the presence of a clear previous history of the disease, the presence perhaps of a splenomegaly, some degree of anaemia and loss of weight and chronic ill health, as well as the occurence of short and mild bouts of fever. Usually the peripheral blood smears are negative, but at times they may be positive if a careful search is made and thick smears prepared.

For this form of disease I would recommend a combined course of quinine and chloroquine (quinine, gr. 10 b.d. for 7 days and gr. 5 b.d. for a further 7 days, with chloroquine, 1 tab. b.d. for 7 days). In addition to these drugs an iron mixture (ferrous sulphate, gr. 5 q.i.d.) and vitamins should be given. Some favour a short course of organic arsenic (0·3 g. N.A.B. once a week for 5 weeks.)

PROPHYLAXIS

In this aspect of the disease progress has been astounding, prophylactic treatment being far more effective than it was a few years ago. This is due to the introduction of several new drugs, chloroquine, paludrine, camoquin and daraprim. Each has its advocates, but the important point to remember is that the tablets should be taken at the stipulated intervals and not too widely spaced.

The prevention of a clinical attack of malaria can be achieved by employing either a causal prophylactic (acting on the pre-erythrocytic forms of the parasites) or a suppressant (which acts on the asexual erythrocytic forms).

I personally advocate chloroquine, which is a safe and powerful suppressant. The dosage required is as follows: For adults—range of 1 to 2 tablets on the same day each week. For children—0-1 years $\frac{1}{8}$ adult dose, 1-3 years $\frac{1}{4}$ adult dose, 3 to 6 years $\frac{1}{2}$ adult dose, 6-9 years $\frac{3}{4}$ adult dose. Over 9 years full adult dose.

Camoquin is another good suppressant. The dosage is 2 tablets (400 mg.) taken as a single dose once a week.

Daraprim is advocated by some as a suppressant. Its dose is: Adults—1 tablet (25 mg.) weekly. Children—5-15 years $\frac{1}{2}$ tablet, under 5 years $\frac{1}{4}$ tablet.

An effective and probably the safest causal prophylactic is proguanil (Paludrine). Provided it is taken daily, it is of definite value in preventing *P. falciparum* infection. The dosage I would recommend is: Adults—100-200 mg. (1-2 tablets) daily. Children—(0-5 years) 50 mg. daily, (6-12) years) 100 mg. daily.

INCREASED FEES PAYABLE UNDER THE WORKMEN'S COMPENSATION ACT : VERHOOGDE GELDE BETAALBAAR INGEVOLGE DIE ONGEVALLEWET

GOVERNMENT NOTICE NO. 2545 OF 10 DECEMBER 1954

Workmen's Compensation Act, No. 30 *of* 1941, *as amended by No.* 27 *of* 1945, *No.* 48 *of* 1947, *No.* 36 *of* 1949, *and No.* 5 *of* 1951.

I, John Henry Lewis, Workmen's Compensation Commissioner, under the powers vested in me by section *seventy-nine* of the Workmen's Compensation Act, 1941, as amended, after consultation with the Medical Association of South Africa, hereby increase the rate of all fees as laid down in the existing scale of fees in Government Notices Nos. 1057 of the 23rd May, 1946, and 182 of the 4th February, 1949, payable in respect of professional services rendered by private medical practitioners, by 25 per cent.

The increase, which will take the form of an addition of 25 per cent. to the total of a doctor's account after assessment in terms of the Medical Handbook, will become effective as from the 1st January, 1955, irrespective of the date of the accident or date of receipt of the relative account, and will not be payable in respect of nursing services, any services (including medical) rendered by hospitals or other institutions or by medical practitioners outside the Union, or in respect of goods supplied by chemists, etc.

The increase will, however, apply in respect of services rendered by registered masseurs and physiotherapists, and by dentists, irrespective of whether they are medical practitioners or not.

Government Notice No. 1784, dated the 21st July, 1950, is hereby withdrawn with effect from the 1st January, 1955.

GOEWERMENTSKENNISGEWING NO. 2545 VAN 10 DESEMBER 1954

Ongevallewet, No. 30 *van* 1941, *soos gewysig by wet No.* 27 *van* 1945, *wet No.* 48 *van* 1947, *wet No.* 36 *van* 1949 *en wet No.* 5 *van* 1951.

Ek, John Henry Lewis, Ongevallekommissaris, maak hierby bekend dat ek, kragtens die bevoegdheid my verleen by artikel *nege-en-sewentig* van die Ongevallewet, 1941, soos gewysig, na beraadslaging met die Mediese Vereniging van Suid-Afrika, hierby die bestaande tariewe vir geneeskundige behandeling vasgestel by Goewermentskennisgewings Nos. 1057 van 23 Mei 1946 en 182 van 4 Februarie 1949, betaalbaar vir professionele dienste deur private mediese praktisyns gelewer, met 25 persent verhoog.

Die verhoging, wat beteken dat 25 persent by die totaal van 'n geneesheer se rekening na berekening ingevolge die Mediese Handboek gevoeg word, tree op 1 Januarie 1955 in werking, ongeag die datum van die ongeval of datum van ontvangs van die betrokke rekening, en is nie betaalbaar vir verpleegdienste nie, nòg vir dienste (geneeskundige dienste inbegrepe) gelewer deur hospitale of ander inrigtings of deur mediese praktisyns buite die Unie, nòg ten opsigte van goedere deur aptekers, ens., gelewer.

Die verhoging sal egter van toepassing wees op dienste gelewer deur geregistreerde masseurs en fisioterapeute, en deur tandartse, ongeag of hulle mediese praktisyns is al dan nie.

Goewermentskennisgewing No. 1784 van 21 Julie 1952 word hierby met ingang van 1 Januarie 1955 ingetrek.

FOURTH SOUTH AFRICAN CONGRESS OF OBSTETRICS AND GYNAECOLOGY

The fourth South African Congress of Obstetrics and Gynaecology will be held in Cape Town from 19 to 22 January 1955. All the meetings unless otherwise notified will take place in the Department of Obstetrics and Gynaecology, Medical School Observatory.

On 19 January Fellows and Members of the Royal College of Obstetricians and Gynaecologists will meet at 3.30 p.m. to discuss (1) the formation of a Regional Council in South Africa and (2) the attitude of Members and Fellows to the South African College of Physicians and Surgeons. At 8 p.m. the Annual General Meeting of the South African Society of Obstetricians and Gynaecologists will take place at which in addition to the election of office bearers and new members there will be further discussion on the attitude of members of the Society to the College of Physicians and Surgeons.

The Programme for the following three days of the Congress is as follows:

Thursday 20 *January*

9.15 a.m. Official opening of the Congress by the Dean of the Faculty of Medicine, University of Cape Town. Professor M. van den Ende.

10.15 a.m. Professor F. J. Ford, Cape Town. 'Resuscitation of the Newborn.'

11.30 a.m. Dr. L. Resnick, Cape Town. 'Foetal Distress.'

12.15 p.m. Dr. J. Black, Johannesburg. Demonstration of a pair of Milne-Murray forceps, designed to make application easier in the R.O.A. position.

2 p.m. Frof. L. J. te Groen, Pretoria. 'Die Uniformiteit van die Baringsmeganisme'.

3—3.45 p.m. Dr. F. Daubenton, Johannesburg. 'Acute Puerperal inversion of the uterus.'

4.15—5 p.m. Dr. T. St. Vincent Buss, Cape Town. 'Virilizing tumours of the ovary.'

Friday 21 *January*

8.00 a.m. Professor J. T. Louw. Wertheim operation at Groote Schuur Hospital.

10.30 a.m. Groote Schuur Hospital Ward C. 10. Discussion on obstetrical cases with Medical complications.

2.15 p.m. Professor O. S. Heyns. 'An Enquiry into the relationship between Stature, Pelvic Capacity and Birth Weight.'

3.00 p.m. Dr. H. G. Rees, Cape Town. 'Anhydramnios—its significance and treatment.'

4.15 p.m. Dr. D. T. Dodds, Port Elizabeth. 'Obstetrics and Gynaecology at the Chicago Lying-in Hospital, University of Chicago.'

Saturday 22 *January*

9.15 a.m. Dr. J. N. de Villiers, Cape Town. 'Afibrinogenaemia in Pregnancy.'

10.00 a.m. Dr. W. H. Muller, Cape Town. 'A Case of Colporrhexis.'

11.15 a.m. Dr. P. F. M. du Toit, Cape Town. 'Cancer of the Uterine Body.'

12.00 noon. Films: 'Colpoperineorrhaphy plus Vaginal Hysterectomy for Prolapse and Enterocoele' (Prof. J. T. Louw). 'Circumcision' (Dr. R. G. Sharp).

2.15 p.m. Dr. D. A. H. du Toit, Cape Town. 'Die Stein-Leventhal Sindroom.'

3.00 p.m. Dr. A. M. Michael, Cape Town. 'Rupture of the Uterus.'

4.15 p.m. Open Discussion on Obstetrical and Gynaecological matters.

Social arrangements during Congress include a banquet for members and their wives at 8 p.m. on Thursday 20 January, a cocktail party by the Mayor and Councillors of Cape Town at the City Hall at 5.30 p.m. on Friday 21 January, and theatre or cinema at 8 p.m. on Saturday 22 January.

MEDIESE GRADE EN DIPLOMAS DEUR DIE UNIVERSITEIT VAN PRETORIA TOEGEKEN

Die volgende kandidate het voldoen aan die vereistes vir die M.B. Ch.B. en ander grade van die Fakulteit vir Geneeskunde van die Universiteit Pretoria:

Graad van Baccalaureus in Geneeskunde en Snykunde

Blokker, Dina Alida.
Booyse, Francois Abraham.
Bornman, Jacobus Johannes.
Botha, Hendrik Stefanus.
Brits, Charlotte Jacoba.
De Bruin, Micha.
De Jager, Josephus Jacobus.
De Klerk, Jacob Jacobus.
De Klerk, Marthinus Christoffel Cornelius.
De Villiers, Pieter Ackermann.
Doussy, Pol Hyacinth Jozef.
Dreyer, Ora.
Duvenhage, Diederick Johannes.
Eiselen, Erhard Heinz Raimund.
Engelbrecht, Jacobus Adriaan.
Engelbrecht, Reniellaume.
Fischer, Peter Ulrich.
Fonternel, Theodorus Ernst.
Hare, Solly Abe.
Hartmann, Horst Emil Wilhelm.
Haumann, Jacob le Roux.
Heydenreich, Johanna Stefana Stoffelina.
Horvath, Tibor.
Hugo, Gabriël Jones.
Jooste, Francois Paulus.
Laubscher, Roedolph Jacobus.
Le Roux, Berté.
Liebenberg, Christiaan Jacobus.
Lohmann, Dietrich.
Lombard, Jan Hendrik Esmeyer.
Louwrens, Cornelius Petrus.
Maartens, Paulus Roald.
Malan, Albertus Johannes Theodorus.
Marais, Melt.
Meyer, Anthonie Christoffel.
Meyer, Jacobus Schabort.
Meyer, Sara Cecilia.
Mieny, Carel Johannes.
Oberholster, Jannie Gerhardus.
Olivier, Johannes Andries.
Pienaar, Henriette.
Pieters, Dirk Cornelius.
Preis, Frants Johan.
Pretorius, David Hermanus Schalk.
Putz, Ingo Herman Joseph Leopold Werner Maria Maximilian.
Reiner, Hans Arnulf.
Reyneke, Johan.
Robbertze, Johannes Hendrik.
Sacchi, Rinaldo.
Scheepers, Coenraad Frederik.
Steyn, André Francois.
Steyn, Johannes MacKenzie Mulder.
Swart, Matilda.
Theron, Frederik.
Theron, Thomas Arnoldus.
Van den Heever, Frans Albertus.
Van der Merwe, Gideon Daniel.
Van Niekerk, Ivan.
Van Rooyen, Peregrine.
Van Tonder, Hendrik Petrus.
Van Zyl, Johannes Abraham.
Van Zyl, Pieter Jacobus.
Venter, Johannes Jacobus.
Viljoen, Frans Jacobus.
Visser, Albert Georg.
Visser, Willem Schalk.
Vorster, Jacobus Francois.
Wassermann, Hercules Petrus.
Welmans, Petrus Gerhardus.
Wolfe, Victor.

Graad van Magister in Geneeskunde

*Meyer, Hendrik. (Dept. Oogheelkunde).
Myburgh, Lambertus Albertus Christiaan. (Dept. Chirurgie).
Pretorius, Christiaan Chrysostomus. (Dept. Obstetrie en Ginekologie).
Rauch, Robert Naylor Laurie. (Dept. Chirurgie).
Van Zinderen Bakker, Rindert. (Dept. Oogheelkunde).

* Dui aan graad met lof.

Diploma in Mediese Radiologie

Esterhuizen, Esterhazy Josias.
Van Rhyn, Johannes Lodewicus van Heerden.

Kliniese Medalje

Vir die beste veterinêre graduandus in Chirurgie, Medisyne en Aansteeklike Siektes:
Basson, Pieter Arnoldus.

Van Riebeeck Goue Medalje

Vir die beste graduandus in Patologie en Histopatologie van die Mondholte in die Fakulteit Tandheelkunde:
Malan, Cilliè.

ELEMENTARY — DR. WATSON

The late Dr. J. H. Watson, who died last year at the age of 100, was a remarkable man, and I was sorry to find a medical author from America, whom I shall not name, comparing his talents unfavourably with those of his friend Mr. Sherlock Holmes. It seems a pity that a medical man should speak thus slightingly of a fellow practitioner, and a London M.D. at that. To compare him with a charlatan like Holmes, who did not even possess a registrable diploma, is almost laughable. Holmes was indeed not completely uneducated; he had a working knowledge of chemistry and anatomy, played the violin, and had been observed to beat the dissecting-room corpses to determine whether bruising could take place after death. These talents, had he applied them, might have fitted him to be a surgeon or perhaps a pathologist, but of the finer points of internal medicine he knew nothing. He was also ignorant of the solar system. That Watson should have allied himself with such a man is curious enough; but it says much for his modesty and nobility of character that he should thereafter have suppressed in his accounts all laudatory mention of his own part in Holmes' investigations, and have given all the credit to his friend.

Apart from his memoirs Watson wrote little, but the *British Medical Journal* for 1887 contains two papers which I take to be his. One is on the use of biniodide of mercury in diphtheria and the other on the opium question in China. Watson was engaged in general practice in London from 1881 to 1939, when despite his age (he was 86) he served with distinction in the armed forces and was twice decorated. In his later years he was famous as a raconteur, but I was never able to discover from him exactly what had become of Holmes. He always said he believed him to be living in retirement somewhere since the death of Moriarty. Watson's health had been poor in his youth, but after his establishment in practice it improved remarkably and his many friends were shocked to hear of his sudden and untimely death during convalescence from an attack of measles. Holmes, of course, was once reported dead in error, and it is just possible that this has happened to Watson too. If so, we may expect a further paper soon on advances in the treatment of diphtheria with biniodide of mercury.

GENEESHEER-GENERAAL VAN DIE UNIE-VERDEDIGINGSMAG

Die aanstelling word bekendgemaak van dr. J. H. Rauch, voormalige Stadsgeneesheer van Germiston, as Geneesheer-Generaal van die leër, met die rang van Brigadier in die Tegniese en Administratiewe Tak.

Jan Hendrik Rauch is 'n gegradueerde van Trinity College, Dublin, waar hy die grade B.A., B.A.O., M.D., Ch.B. in 1923 verwerf het. In 1932 verkry hy die graad D.P.H. aan die Witwatersrandse Universiteit, en in 1939 die D.T.M. & H. Sedert 1932 is hy met gesondheidsadministrasie betrokke, eers as Assistent-stadsgeneesheer, Johannesburg, en in 1935 is hy aangestel as die eerste voltydse Stadsgeneesheer van Germiston. Hier het hy gesondheidsdienste vir die stad georganiseer, en was verantwoordelik vir die oprigting van poliklinieke vir beide blankes en nie-blankes.

Dr. Rauch het besondere ondervinding opgedoen weens sy aanstelling sedert 1935 as lugvaartgesondheidsbeampte vir die Randse Lughawe en later vir Palmietfontein. 'n Aantal verslae oor die geskiktheid van landingsterreine in verskillende dele van die vasteland van Afrika vanaf die gesondheidsoogpunt is deur hom opgestel.

Dr. Rauch staan welbekend in Transvaalse volksgesondheidskringe waar hy dikwels 'n leidende rol gespeel het. Hy was o.a. Voorsitter van die Witwatersrandse en Pretoriase Gesondheids-Konsulterende Komitee, lid van die Nasionale Gesondheidsraad, lid van die Raad van die Suid-Afrikaanse Buro vir Standaarde, en Voorsitter van die Tegniese Subkomitee vir Terapeutiese Stowwe. Daarbenewens is hy lid van die Suid-Afrikaanse Akademie vir Wetenskap en Kuns, en eksterne eksaminator vir die Koninklike Sanitêre Instituut en vir die Universiteit Potchefstroom. Hy is dan ook 'n lid van die Mediese Vereniging.

In 1951 het hy 'n uitgebreide studiereis in Europa en Noord-Afrika onderneem, tydens sy afvaardiging na 'n sanitêre kongres in Engeland.

Sy eggenote, dr. J. V. Pietersen, is tans senior lektrise in Volksgesondheid aan die Universiteit Witwatersrand.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following contributions to the Benevolent Fund during August—November 1954, are gratefully acknowledged.

Votive Cards in Memory of

Mrs. van Heerde *by* Dr. J. P. Immelman.
Mrs. Mollie Smart *by* Dr. L. E. Miller.
Mr. D. M. Buchanan *by* Dr. & Mrs. A. W. Sichel.
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Mrs. R. D. Pilkington-Jordan *by* Dr. & Mrs. A. W. Sichel and Dr. Vernon Brink.
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Dr. F. Brett-Young *by* Dr. L. B. Goldschmidt.
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Dr. Malcolm C. G. Dods *by* The Pensions Office, Johannesburg.
Dr. J. Pasvol *by* The Matron and Sisters, Victoria Hospital, Wynberg.
Dr. J. Duff Scott *by* Dr. W. C. McDavid and Dr. Cyril Frost.
Mr. P. D. Rousseau *by* Dr. & Mrs. J. S. du Toit.
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Mrs. I. M. Harwood-Nash *by* Dr. & Mrs. K. M. Scott.

Total Amount received from Votive Cards £70 10 0

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Ivan, son of Dr. Tockar *by* Drs. Arnold Katz, B. Chorn and P. Jenkin.
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Dr. Maurice Haifer *by* Dr. J. Edelstein and Dr. Les Blum (Dentist).
A Member of the family of Dr. M. Preiss *by* Dr. J. Rudolph.
The Family of Dr. D. Anderson *by* Dr. N. Keevy.
'Anonymous' *by* Dr. Patricia Massey.
Dr. Dyke *by* Dr. L. M. Oosthuizen.
Jennefer *by* Drs. Margaret Orford and F. W. Roberts.

	£	s.	d.
Total Amount received from Services Rendered ..	£65	12	0
DONATIONS:	£	s.	d.
'Anonymous'	4	4	0
Special bequest estate late Dr. A. M. Pollock ..	500	0	0
Donations from Members of Cape Western Branch (Collection Box)	8	10	0
39th S.A. Medical Congress held at Port Elizabeth	12	4	7
Decor Flowers and Gifts, Port Elizabeth ..	3	0	0
Proceeds from the Campbell Cup Golf Competition held at Paarl	2	18	3
Dr. J. Henson	3	3	0
Dr. S. Goldberg ('The Physician's Prayer') ..	1	1	0
Dr. J. Grove ('The Physician's Prayer')	1	0	0
Dr. D. Goddard ('The Physician's Prayer') ..		10	0
Dr. H. W. B. Spicer ('The Physician's Prayer')		10	0
Dr. B. Morrison ('The Physician's Prayer') ..		5	6
Mr. J. A. du Toit ('The Physician's Prayer') ..		1	0
'Anonymous' ('The Physician's Prayer') ..		1	0
Dr. W. P. Steenkamp, Jnr. ('The Physician's Prayer')		10	6
Dr. H. Bernstein ('The Physician's Prayer') ..		1	0
Dr. G. Ismay ('The Physician's Prayer') ..		1	0
Dr. S. P. Ferreira ('The Physician's Prayer') ..		3	0
Dr. F. A. Dreyer ('The Physician's Prayer') ..		1	0
Dr. G. Pieterse ('The Physician's Prayer') ..		1	6
Dr. W. Norman Taylor ('The Physician's Prayer')	1	1	0
American College of Chest Physicians (Northern Chapter)	5	5	0
Donations ex Mannequin Parade and Ball, Southern Transvaal Branch	1,400	0	0
Proceeds from the Jones Phillipson Golf-Cup Competition	1	11	0
Dr. H. Saacks		10	6
Dr. P. A. Euvrard		5	0
Dr. C. Slome		10	6
Total	£2,083	11	4

PASSING EVENTS : IN DIE VERBYGAAN

The South African Medical and Dental Council. The Minister of Health has approved an amendment of the rules made by the South African Medical and Dental Council which adds the following to the list of additional qualifications registrable by medical practitioners: Royal College of Surgeons of England: Fellow of the Faculty of Anaesthetists (F.F.A., R.C.S. Eng.).

* * *

The Society of Thoracic Surgeons of South Africa, a group of the Medical Association of South Africa, the constitution of which was approved by Federal Council in October, has elected the following office-bearers for the first year: Mr. L. Fatti (Johannesburg), Chairman; Mr. W. Phillips (Cape Town), Vice-Chairman; Mr. G. R. Crawshaw (Johannesburg), Hon. Secretary and Treasurer.

* * *

Dr. Irene E. Kenmuir, B.A., M.B., Ch.B. (U.C.T.), D.C.H. (R.C.P. & S.), has commenced practice at 'Ranfurly', Kenilworth Road, Kenilworth, Cape. Telephone: 7–1875.

Dr. Kenmuir recently spent 3 years overseas where she studied at the Great Ormond Street Hospital for Sick Children, and afterwards obtained the Diploma in Child Health and engaged in a general practice in England for 2 years. Dr. Kenmuir has recently been undertaking work as *locum tenens* in various parts of South Africa.

* * *

Dr. Gordon James Key, Superintendent of the Valkenberg Mental Hospital for more than 34 years, retired from that position on 28 December. He has also resigned as Lecturer in Charge of the Department of Psychiatry at the University of Cape Town. After a short holiday, Dr. Key will go into private practice in Cape Town.

Presentations from the European and non-European Staff were made to him before he left the Valkenberg Hospital.

* * *

Dr. J. H. (Jannie) Louw, M.B., Ch.B., M.Ch., Dean of the Medical Residence of the University of Cape Town, has been appointed Associate Professor of Surgery at the University. Dr. Louw, who is 39, is a surgeon at Groote Schuur Hospital and head of the Children's Surgical Unit there.

* * *

The 7th Congress of the Medical Women's International Association took place at Gardone Riviera, Italy, on 15-21 September 1954. There were 300 delegates representing 22 countries in 5 continents.

The official inauguration took place at a brilliant reception given by the Mayor of Brescia in the Salon Vanvitellien, Brescia, in the presence of the municipal authorities and the medical profession.

The Congress members were then invited to the new 'Opitai Civil' at Brescia, which is of great interest, both from the point of view of architecture and its modern equipment.

The scientific sessions of the Congress were held in the Assembly Room of the Grand Hotel Gardone, where a service of simultaneous interpretation into Italian, French and English had been installed.

The main theme of the Congress was *The Menopause* with *The Problems of Very Early Marriage* as a subsidiary subject. There was also a session devoted to papers given by eminent Italian women doctors.

The Congress members were taken on a tour of Lake Garda, and were received at Riva by the local authorities, where they visited medical institutions; they also visited the hospital of the Italian Red Cross at Malcesine, where they were received by the President of the Italian Red Cross, Prof. Longhena.

The orchestra of the Scala, Milan, under the direction of M. Votto, gave a memorable concert at the Vittoriale in honour of the Congress.

Dr. M. Yolanda Tosono Dalai, of Milan, was elected the new President of the International Association, and it was decided that the next meeting of Congress, in 1958, should be in London.

* * *

South African Society of Medical Women. Medical Women who have not returned the Society's questionnaire are asked to do so as soon as possible.

The Society is endeavouring to compile statistics which will be of importance in supporting negotiations now being entered into.

REVIEWS OF BOOKS : BOEKRESENSIES

CLINICAL ORTHOPAEDICS

Clinical Orthopaedics, No. 3. Edited by Anthony F. DePalma, with the assistance of the Associate Editors and the Board of Advisory Editors. (Pp. 230 + viii, with illustrations. 60s.) Philadelphia; London; Montreal: J. B. Lippincott Company. 1954.

Contents: Part I. Soft-Tissue Conditions Around Joints. 1. A Review of the Surgical Treatment of Congenital Dislocation, Recurrent Dislocation, or Slipping Patella. 2. Sprains. 3. Injuries to the Ligaments of the Knee. 4. Cysts of the Semilunar Cartilages of the Knee. 5. Traumatic Lesions of Menisci. 6. Injuries of the Medial Meniscus. 7. Recurrent Dislocation of the Patella. 8. Treatment of Dislocation of the Patella. 9. Avulsion of the Hamstring Tendons from the Ischial Tuberosity: Report of Case. 10. Bicipital Tenosynovitis. 11. Recurrent Anterior Dislocation of the Shoulder Joint. 12. Ruptures of the Rotator Cuff. 13. Injuries of the Ligamentous and Associated Structures about the Hip Joint. 14. Sprains and Ruptures of Ligaments of the Ankle Joint. 15. The Pellegrini-Stieda Para-articular Calcification. *Part II. General Orthopaedics.* 16. Giant-Cell Tumour of Bone and its Differential Diagnosis. 17. Step-Cut Knee Fusion with Medullary Fixation. 18. Closed Medullary Pinning of Celles' Fracture. 19. A Comparative Clinical Study of Autogenous and Frozen Homogenous Bone in Grafting Procedures. 20. The Use of Tubadil (Repository Injection of Tubocararine) in Active Back Strain. 21. Fatigue Fracture of the Shaft of the Femur. 22. Vascular Relations of the Ankle and their Clinical Significance. 23. Treatment of Femoral-Neck Fractures. Index.

This volume contains 23 separate articles on a wide variety of more or less unrelated orthopaedic subjects, by a total of 31 authors. It is therefore inevitable that the style and the scientific standard of the articles must vary as much as in any ordinary journal of surgery. In fact, this book consists of nothing more than a fairly average edition of the Journal of Bone and Joint Surgery, in a much more expensive form. It therefore has no resemblance to a text-book and is not intended for students or for practitioners as a reference book on Orthopaedics. The title is somewhat misleading in this respect.

It contains a number of articles on original work done by the authors. Much of this work is naturally still quite unproved, and many of the opinions expressed represent the views held by one or possibly two authors only, and would certainly not be universally or even widely accepted.

Article No. 9 is a report on a single case of rupture of the hamstring tendons which the two authors declare is an extremely rare injury. Such a report would be more in place in a journal than in a book.

In an article on the surgery of recurrent dislocation of the patella, the author declares, 'I have not hesitated to employ pin fixation through both tibia and femur—until healing is well established'. No details of this 'pin fixation' are given, and the whole article consists of enumerating all of the dozens of unsatisfactory operations that have been tried for this condition, with comments on how unsatisfactory they nearly all are, but without describing a single one or offering anything better.

Article No. 10 deals well with the pathology and treatment of bicipital tenosynovitis, a condition which, as the authors infer, is so often missed as the cause of a painful shoulder.

Article No. 11 describes the Magnuson operation for recurrent dislocation of the shoulder, which the authors considered the best procedure to adopt, although it produced a failure rate of 10·5%. On the cover of the 'Yearbook of Orthopaedics and Traumatic Surgery' of a few years ago, one of the 'spot' questions was 'what is the Johannesburg Staple operation'? It is an operation to cure recurrent dislocation of the shoulder, and although accurate figures are not known, I believe that the failure rate is less than 10·5%.

Article No. 22 is an excellent study of the vascular relations of the ankle joint, which explains adequately a number of complications of sprains and fractures in this region, such as persistent

pain (due to trauma and resulting spasm of the dorsalis pedis artery), avascular necrosis of the talus, and the ever-recurring non-union of the medial melleolus. The upper and lower photographs on page 199 are described the wrong way round.

The Editor-in-Chief states in his foreword: *Clinical Orthopaedics* is designed for the publication of original articles offering significant contributions to the advancement of surgical knowledge. About one half of the articles may qualify for this description. For the full-time orthopaedist, or for any surgeon whose work embraces a large proportion of traumatic work, this book must be read if he wishes to keep abreast of everything that is new in the field of orthopaedics today. The total amount of new literature in English available on orthopaedics is certainly very small compared with what the average specialist physician or general surgeon has to read through. Unfortunately many of the best articles on purely orthopaedic subjects are found singly and scattered about in the dozens of journals of general surgery, and even in *Surgery, Gynaecology and Obstetrics*.

This collection of articles entitled *Clinical Orthopaedics* is therefore a most welcome publication. Hi. R.

ALCOHOLISM

Alcoholism. Jackson A. Smith, M.D. (Pp. 72+vii. 24s.) Philadelphia; London; Montreal: J. B. Lippincott Co. 1954.

Contents: 1. Behaviour Leading to the Diagnosis of Chronic Alcoholism. 2. Psychotherapy as Applied to the Chronic Alcoholic. 3. Delirium Tremens. 4. Antabuse. 5. Other Types of Treatment. 6. The Treatment of Alcoholism in General Practice. Index.

This book presents in a most readable way the essentials of alcoholism for the hospital intern in contact with the problem, as well as for the medical man in active practice. The author has shorn the subject of all unnecessary fads and misconceptions in the approach, the concept, and the treatment.

The author deals adequately with his subject, basing it on an obviously vast practical experience and on an understanding of the complexities of the problem.

The reading of the book is not time-consuming and, being pocket-size, it can easily be made available at all times. For those particularly interested in the subject, there is a most useful list of references. B.S.

HEALTH IN A THOUSAND INFANTS

A Thousand Families in Newcastle-upon-Tyne: An Approach to the Study of Health and Illness in Children. By James Spence, W. S. Walton, F. J. W. Miller and S. D. M. Court. (Pp. 217+xiv, with 8 figures. 56 tables and 7 plates. 10s. 6d.) London; New York; Toronto: Oxford University Press. 1954.

Contents: Part I. 1. The Present Investigation. 2. Our Families. 3. The City of Newcastle-upon-Tyne. 4. The Development of Medical Provision for Infants and Children. *Part II.* 5. The General Pattern of Illness in the First Year. 6. Causes of Death in the First Year. 7. Congenital Abnormalities. 8. Respiratory Disease: (*a*) Acute Respiratory Disease; (*b*) Whooping-Cough; (*c*) The Family Setting and Medical Care in Respiratory Disease. 9. Tuberculosis in the First Year of Life. 10. Diarrhoea and Vomiting. 11. The Infectious Fevers. 12. Staphylococcal Infection. 13. Some Infrequent yet Serious Diseases. 14. Convulsions. 15. Circumcision. 16. Accidents. *Part III.* 17. Housing in Newcastle-upon-Tyne, 1947-48. 18. The Mother's Care of the Child. 19. Infant-Feeding. 20. Mastitis and Breast Abscess. 21. The Use of the Dummy. 22. Prematurity. 23. Illegitimacy. 24. Problem Families. 25. The Medical Care of the Children: (*a*) The Family Doctor; (*b*) The Hospital; (*c*) The Child-Welfare Centre. *Part IV.* 26. Summary. 27. Conclusions and Suggestions. *Part V.* Appendices. Index.

This record of a longitudinal study of the health of a thousand infants viewed against the background of their respective families is regarded by his colleagues as a memorial to their leader and companion, Sir James Spence.

The results illustrate the value of co-operation between Local Authority, University Teaching Department and General Practitioners in research, and their approach to the parents is expressed in the remark that 'we tried to remain sensitive to the privilege of access to the homes of our families'. All the 1,142 babies born in Newcastle in May and June 1947 were taken as the sample to be studied and only 4 families failed to co-operate. After deductions for babies who died or who were removed 967 infants remained, who were followed throughout the year.

Only one-fifth of the infants escaped illness before their first birthday. The remainder had an average of 2 significant illnesses each during the year. Over 80% of these morbidities were due to infections, mainly respiratory. The epidemiology of respiratory, gastro-intestinal, staphylococcal and tuberculotic infections, as well as that of the infectious fevers and accidents, revealed the importance of seeing the infant in its family context.

In the discussion on causes of infantile mortality, stress is laid upon multiple etiology. Of the 44 infant deaths which occurred, no less than 15 were considered avoidable.

The importance of the mother's care of the child as a factor in the environment is summarized by the statement that 'the mother stands out as the corner-stone of the family structure, and our experience confirms that in all sections of society she remains the chief guardian of child welfare, a fact which is sometimes in danger of being forgotten'.

The use of family doctors, hospitals and infant-welfare clinics was correlated with social and health factors and provides interesting social and medical data.

The authors finally make a number of recommendations directed towards the improvement of medical education in paediatrics and family practice, and stress the value of integration between preventive and curative services.

This book should be read by all family doctors, paediatricians, medical educators and those involved in public-health practice. H.T.P.

CLEFT PALATE AND SPEECH

Cleft Palate and Speech. By Muriel E. Morley, B.Sc., F.C.S.T. Third Edition. (Pp. 173+xx, with 64 figures. 17s. 6d.) Edinburgh and London: E. & S. Livingstone Limited. 1954.

Contents: 1. Development of the Face and Mouth in Congenital Clefts of Lip and Palate. 2. The Normal Palate. 3. An Outline Story of the Development of Cleft Palate Surgery. 4. Problems Associated with Cleft Palate: Feeding and Speech. 5. Typical Defects in Cleft Palate Speech. 6. Treatment I. General Considerations. 7. Further Suggestions for Treatment. 8. Case Histories. Index.

This book, as its title suggests, deals mainly with speech training after cleft-palate operations, but assessment of speech after different kinds of operation have led the author to begin with an excellent résumé of the history, embryology and anatomy of cleft lip and palate, and a clear up-to-the-minute exposition of the various types of cleft-palate operation.

It is an essential book for the palate surgery: it shows how dependent the surgeon is on the speech therapist for his final results and how important it is for the surgeon to provide the patient with a palate of sufficient length and mobility. Too often has the surgeon blamed his results on 'faulty speech patterns' where the cause has actually been inadequate surgery. D.S.D.

BOOKS RECEIVED : BOEKE ONTVANG

The Cerebrospinal Fluid. By S. Lups, and A. M. F. H. Haan. (Pp. 350 +xv, with illustrations. 52s. 6d.) Amsterdam; Houston; London and New York: Elsevier Publishing Company. 1954.

Brain Mechanisms and Consciousness. A Symposium Organized by the Council for International Organizations of Medical Sciences. (Pp. 556 + xv, with illustrations. 42s.) Oxford: Blackwell Scientific Publications. 1954.

Living with the Dead. By D. J. Coetzee. 2nd. Ed. (Pp. 89. 10s.) Published by the Author, Observatory, Cape.

Babies and Young Children: Feeding, Management and Care. By Ronald S. Illingworth and C. M. Illingworth. (Pp. 360 + vii, with 24 plates and 50 text-figures.) London: J. & A. Churchill Limited. 1954.

Atlas of Congenital Cardiac Disease. By Maude E. Abbott. (Pp. 62 +x, with illustrations.) New York: American Heart Association, Inc. 1936.

Three Men: An Experiment in the Biography of Emotion. By Jean Evans. (Pp. 297+xviii. 15s.) London: Victor Gollancz Limited.

Fat Metabolism. A Symposium on the Clinical and Biochemical Aspects of Fat Utilization in Health and Disease. Edited by V. A. Najjar. (Pp. 185+viii, with illustrations.) Baltimore: The Johns Hopkins Press. 1954.

Haematological Technique for Medical Laboratory Technicians and Medical Students. By E. M. Darmady and S. G. T. Davenport. (Pp. 197+viii, with 4 coloured plates and 23 text figures. 18s.) London: J. & A. Churchill Limited. 1954.

CORRESPONDENCE : BRIEWERUBRIEK

THE DURBAN MEDICAL SCHOOL

To the Editor: I should like to assure my friend Dr. Louis Freed and others who are interested that those now responsible for the Durban Medical School are not unmindful of the debt owed to the early exponents of the idea of such a School. I am the proud possessor of a copy of the 'Loram Report' autographed and given to me by the late Dr. Loram himself, in 1928, together with an expression of the hope that I, then a newly qualified practitioner commencing medical missionary work in the Tugela Valley, would one day be on the staff of a Medical School for Natives. Our Board of Faculty has already discussed proposals for perpetuating, within the Medical School, the memory of its progenitors.

Among these I should like to mention one not mentioned by Dr. Freed—the late Dr. Sam Campbell of Durban, who was to have been a member of the Loram Committee. He fell seriously ill and died early in 1926, but not before he had done some important preparatory work among his colleagues in Durban who later gave evidence to the Committee. By a happy circumstance, his son, Dr. George Campbell, is now Chairman of the Council of the University of Natal, which is responsible for the Durban Medical School.

Dr. Sam Campbell was in favour of Durban as the site, but the Loram Committee recommended Johannesburg. At that time the University Senates both at Cape Town and at Johannesburg were averse to non-segregated classes, and had the Loram Committee's recommendations been carried out the School in Johannesburg would have been 'segregated'. I judge, from some current comments I have heard, that many of my colleagues in the Witwatersrand Medical School are not aware of this bit of history.

Professor R. A. Dart is the only surviving member of the Loram Committee. He was invited to be the external examiner in the first professional examination held in this School—in November, 1953. Unfortunately circumstances beyond his control prevented him from coming, but he came this year, and I had the pleasure and honour of showing him the fulfilment of a vision which he and I had shared with Dr. Loram 26 years ago. We then envisaged a School costing about £65,000. The present School will have cost nearly £500,000.

The correct appellation is not the 'Non-European' Medical School, but the Durban Medical School. We expect to have European post-graduates students soon.

G. W. Gale
Dean, Durban Medical School

University of Natal
Durban
15 December 1954

THE SPECIALIST REGISTER

To the Editor: In decrying the disabilities of the competent medical practitioner *vis à vis* the specialist register, your correspondent Pittalus displays an appalling ignorance of the true facts of the position. This question was the main issue in the appeal by the Council against a judgment of Mr. Justice de Villiers in the case of Shapiro v S.A. Medical & Dental Council.

Dealing with the arguments addressed to the Court by Mr. Findlay, Q.C., in support of the Council's appeal, Mr. Justice Murray, with the concurrence of his fellow judges, Ramsbottom J. and Malan J., commented as follows:

'It was argued that the Council did not prohibit a medical practitioner from *de facto* practising as a specialist by concentrating or restricting his activities to one particular branch of medical function: only his 'holding himself out' to be a specialist in such branch was regarded as improper. This argument does not appear to be well founded.

'Until the statute defines the term "specialist" either in express terms or by deputing to the Council the power to define it, the Council is not entitled, in my view, to define the term in such a way as to make itself the sole arbiter of what is a specialist, and of what a specialist may or may not do in a particular field of specialist activity. In the absence of an authorised limitation of the term, a medical practitioner who is *in fact* a specialist—though not registered by the Council as one—cannot be said to claim the possession of a competence which he does not possess, provided (it may safely be assumed) that he does not misrepresent himself to have been recognised by the Council as a specialist.

'The Council, as stated by Mr. Findlay on its behalf, disclaims any intention of preventing a medical practitioner from performing any professional function which his registration permits: the contention is that the mischief is one of "holding out", in the same way as certain of the earlier ethical rules prohibit advertising and control name plates, letterheads etc. The present case does not concern any improper methods adopted by a medical practitioner (not registered as a specialist with the Council) in making it known that he specialises in a particular field. As the learned Judge points out, it is inevitable that a medical practitioner who proposes to restrict his practice to the treatment of a particular disease or the exercise of a particular branch of the profession must inevitably indicate both to the public and to the profession that he specialises therein. But (as above stated) he does not thereby claim a competence which he in truth does not possess.'

In 1954, certain sections of the Medical, Dental & Pharmacy Act were amended with the purpose of restoring the position which had previously existed under the Medical Council's rules and which had been declared *ultra vires* by the Courts. Actually, the legislature went even further than was ostensibly contemplated by the Council itself. Despite the very lucid exposition by the Supreme Court judges of the issues involved in defining what is a specialist, Section 33(3) of the Act was amended to read as follows:

'No registered person shall take, use or publish any name, title, description or symbol indicating or calculated to lead persons to infer that he possesses any professional qualification which is not shown in the register in connection with his name, *nor shall any registered person practice as a specialist or hold himself out to be a specialist unless his speciality has been registered as prescribed by the Council.*'

All ambiguity has thus been removed. In terms of our law a 'specialist' now means a person who has been registered as such by the Council and any other practitioner—whatever his competence in a special field—who 'practises as a specialist' or 'holds himself out to be a specialist' without being registered as such by the Council commits an offence for which he may be apprehended by the police like any unlicensed quack.

All the things which Pittalus naively assumes that the competent general practitioner is entitled to do if he so wishes are in fact now illegal. It is time that Pittalus realized that the new law and the illegal regulations—now validated—which preceded it, have taken away fundamental rights which medical men in most civilized countries of the world enjoy without restriction as members of an honourable profession; that the profession in this country has been divided by law into two classes with different rights and privileges; and that unless we oppose with the utmost resolution those who seek by restrictive rule and regulation to maintain and extend the present iniquitous system of professional segregation based on academic snobbery and limitation of opportunity, all the cherished traditional liberties upon which the full development and expression of the doctor's personality depend will irretrievably be lost.

Maurice Shapiro

P.O. Box 9326
Johannesburg
18 December 1954

'SWIMMER'S ITCH IN SOUTH AFRICA' S. AFR. MED. J., 1954 **28**, 1062

To the Editor: I think it only fair to the photographer that your readers should know that the horrible reproduction accompanying the above mentioned article bears no relationship whatever to the excellent original you were provided with, and of course, does not even faintly resemble the lesions seen in 'Swimmer's Itch'.

I apologize to your readers who may mistake it for a lunar landscape or a scene in drought-stricken Texas. I would like them to know that for reasons, known only to yourself, you made no use of the Institute's offer to pay for special art paper on which reasonably clear reproduction can be made.

Botha de Meillon

S.A. Institute for Medical Research
P.O. Box 1038
Johannesburg
13 December 1954

The picture referred to will be found on p. 1063 of the issue of 11 December 1954. *Editor.*